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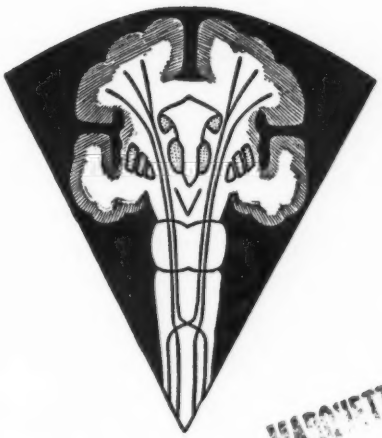

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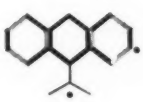

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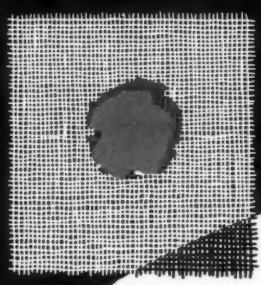

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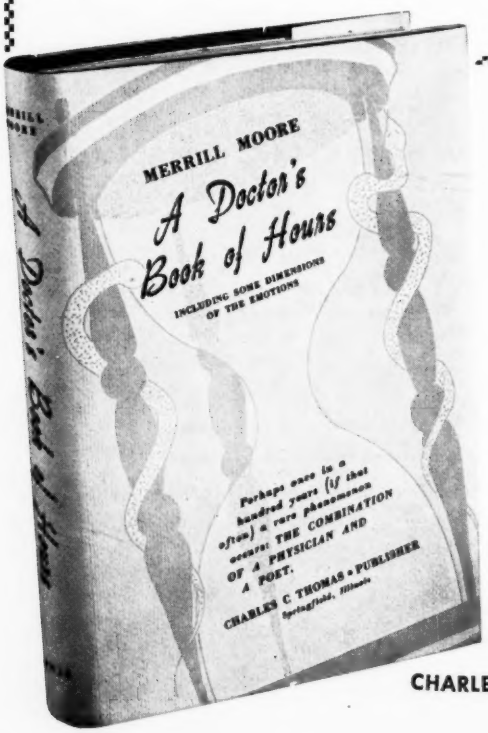
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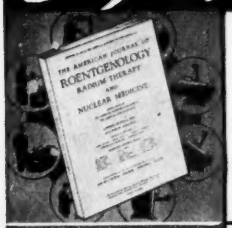

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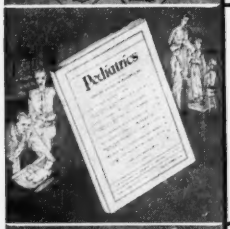

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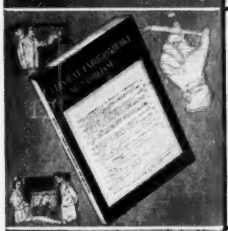

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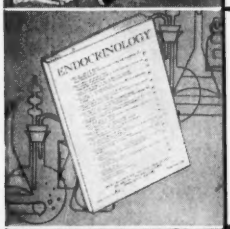

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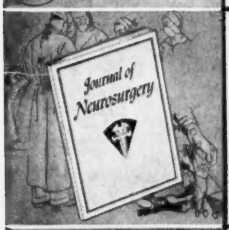

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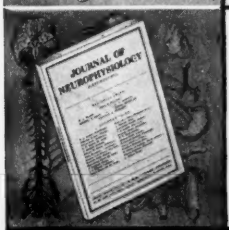

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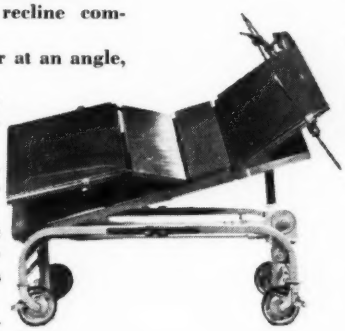

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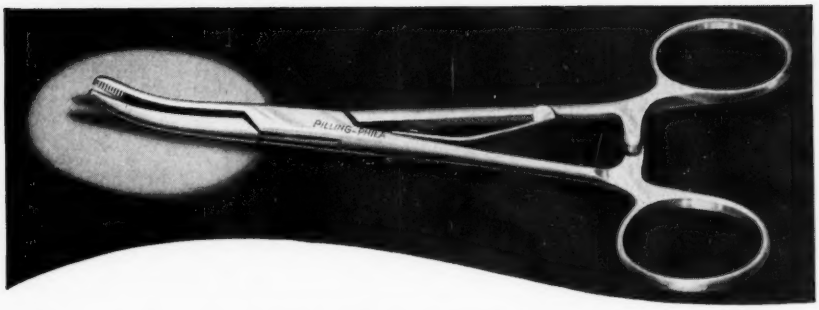

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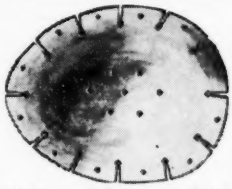

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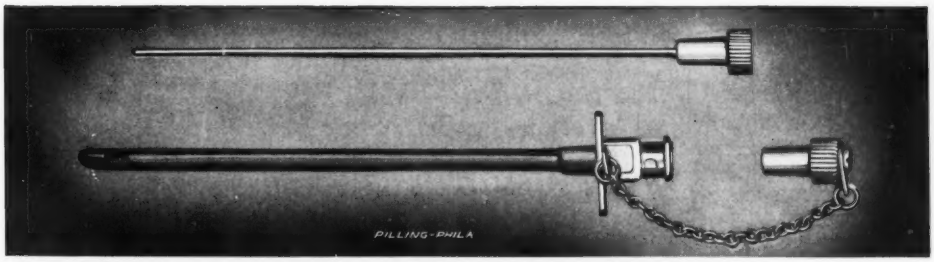

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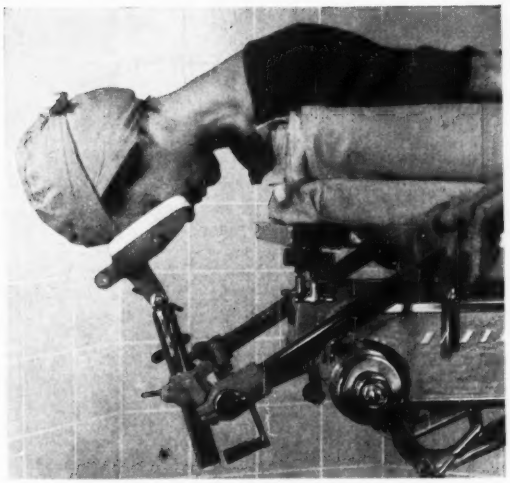

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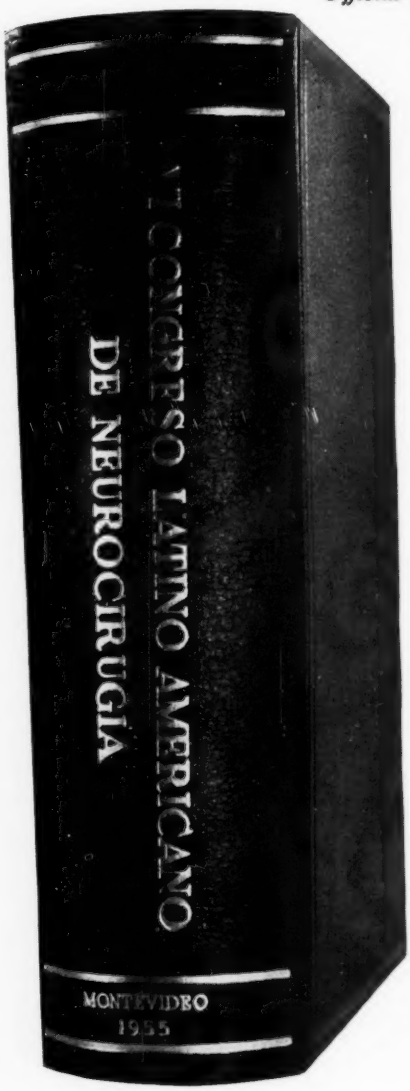

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HYDROENCEPHALODYSPLASIA

AN ANATOMICOCLINICAL PICTURE

J. A. PICAZA, M.D.,* G. CARDELLE, M.D., AND R. MARTIN JIMENEZ, M.D.
Hospital Municipal de Infancia, Havana, Cuba

(Received for publication March 24, 1955)

IN THIS study we are presenting an anatomicoclinical picture which results from increased intracranial pressure in patients with extensive dysplasia of the brain.

Wilson's[26] use of the generic term *encephalo-dysplasia*, a "comprehensive term for the multitudinous aberrant faults of development to which the brain is subject," is inclusive of all brain malformations. Certain malformations present anatomicoclinical and surgical reasons for consideration as a separate group, and we have therefore suggested the term *hydroencephalodysplasia* as applying to those patients who show signs of increased intracranial pressure at any period in their development.

For diagnostic purposes hydrocephalus in infants was differentiated according to the nature of the lesion, and the conclusion was reached that porencephaly should be considered as a related anomaly. Porencephaly has not been well defined. Heschel[13] considered porencephaly a cerebral defect that allowed communication between the subarachnoid space and the ventricles. However contemporary authors[7,8,15,18–20] are inclined to consider this condition as any malformation characterized by extensive loss of brain tissue whether in communication or not with the subarachnoid space or the ventricles. Intracranial pressure has been reported in patients who at varying times in life manifest symptoms of a porencephalic cyst.[9,16] Cruveilhier[4] noted that patients with a large part of the brain missing could be either microcephalic or hydrocephalic. The latter condition he called "hydro-anencephaly" and felt certain that the "atrophy" was not the result of the pressure of fluids. Similar instances in which the cerebral hemispheres were missing have been reported by others.[2,6,10,12,14,17,23,25]

External hydrocephalus, that is, the accumulation of cerebrospinal fluid under increased pressure on the external surface of the brain, is unusual as a primary condition. Hygromas and subdural hematomas have been described under this heading in ancient times. According to Dandy,[6] external hydrocephalus may occur as follows: In cases of communicating internal hydrocephalus, the cerebrospinal fluid may so distend the cisterns that they bulge and compress the brain, and even the arachnoid membrane may break and allow the fluid to escape to the subdural space where it cannot be ab-

* I No. 506, Vedado, Havana, Cuba.

sorbed, thus causing gradually increasing compression of the brain. This seldom happens spontaneously although it is a not infrequent complication of operations for hydrocephalus when the arachnoid membrane is opened or the cortex is divided.

In a series of 135 consecutive cases of hydrocephalus we have not found a single instance of primary external hydrocephalus as described by Dandy. Furthermore, wherever there was an accumulation of fluid over the surface of the brain there was extensive encephalodysplasia. This condition we have named *hydroencephalodysplasia*. Cushing (1928) stated that true external hydrocephalus may be associated with congenital anomalies. Probably his illustration in *Osler's Modern Medicine* (p. 257)[5] is of this condition.

FIG. 1. Hydranencephalic baby. Appearance is not unlike that seen in infantile internal hydrocephalus.

In a previous study of 50 cases of hydrocephalus, the authors[21] found 11 such malformations, then designated "hydrodysplasia." These cases are included in the present study. The criteria are the same as in the previous study but the designation, *hydroencephalodysplasia*, (hydro, water; enkefalo, head content; dysplasia, maldevelopment) is considered more applicable to the condition. Twelve cases have been added, making a total of 23 cases of hydroencephalodysplasia in this study of 135 hydrocephalics.

CLINICAL SYMPTOMS

The ages of the patients ranged from 23 days to 2 years; 17 of the patients were males, 6 females; 21 were white, 2 mulatto (white and negro). Although the proportion of negroes in Cuba is as high as 20 per cent in certain areas there were no pure negroes in any of the groups.

GENERAL SYMPTOMS

The two most outstanding and constant findings were deformity of the head and psychomotor arrest. These symptoms were present to some degree in all patients. The increase in size of the head was similar to that seen in hydrocephalus and measurements varied from 41 cm. in circumference at 1 month to 56 cm. at 4 months. The fontanels bulged slightly and the shape of the head was not unlike that seen in congenital hydrocephalus (Fig. 1). There was one example of turricephaly which was probably the result of a certain amount of premature synostosis of the parietotemporal and lambdoid sutures. One patient was definitely microcephalic with a head circumference of 41 cm. at 2 years of age. Probably, increased cerebrospinal fluid pressure was present after completion of the premature closure of the cranial sutures.

In a previous review,[21] we mentioned the phenomenon of transillumination of the skull or fontanels by a strong beam of light. More recently Hamby[12] has described and beautifully illustrated this phenomenon. Trans-

FIG. 2. Transillumination of the skull.

illumination of the skull was first noted by us in an accidental observation of a patient in the sunshine. The best procedure for demonstration is to place the patient in a dark room and focus a strong beam of light on the skull. This can be demonstrated in advanced hydrocephalus and is not pathognomonic of hydroencephalodysplasia (Fig. 2).

The physical development of a baby affected with hydroencephalodysplasia is normal externally but, on the other hand, relationship to environment is very limited. This accounts for the characteristic "indifferent facies" which becomes more remarkable as the patient grows older. The baby exhibits very little spontaneous behavior, will not follow objects with his eyes and shows little or no facial expression. The only reaction is by such primitive behavior as crying. In patients with less extensive malformations these symptoms are present though not so noticeable. In 6 patients crying was continuous for a few days after birth; in 5, hyperirritability was evident for several months after birth and expressed by a tendency to cry from any stimulus.

Five patients were cyanotic at birth. In the histories of 2 were symptoms of miscarriage at the sixth month of pregnancy; in 1 there was a prolonged delivery; in the remaining 2 there were no abnormal conditions. Cyanosis may be the result of the malformation rather than the cause, or the result of a nervous system inadequate to maintain life.

OPHTHALMOLOGICAL SYMPTOMS

The majority of the patients exhibited some type of ophthalmic impairment. Strabismus was the result of complete third nerve palsy in 2 patients but in 3 others there was bilateral external strabismus. Spontaneous nystagmus, mydriasis, complete absence of photomotor reflex, and probably complete amaurosis were symptoms of the patients with severe optic atrophy.

Funduscopic studies revealed simple optic atrophy in half the patients with no characteristic findings. In one instance this condition was associated

TABLE 1

Incidence of clinical symptoms in hydroencephalodysplasia

General Symptoms	No. of Cases	Neurological Symptoms	No. of Cases	Ophthalmological Symptoms	No. of Cases
Deformity of head	21	Psychomotor arrest	21	Strabismus	3
a. Macrocephalic 19		Convulsions	10	Spontaneous nystagmus	3
b. Microcephalic 1		a. Generalized 9		Loss of photomotor reflex	3
c. Turricephalic 1		b. Jacksonian 1		Mydriasis	2
Transillumination of skull	6	Rigidity	6	Microphthalmus, loss of corneal reflex	1
Indifferent behavior	13	Generalized hyperreflexia	4	Fundus	11
Continuous crying	6	Hemiplegia	3	a. Optic atrophy 10	
Hyperirritability	5	Loss of sucking reflex	1	b. Abnormal distribution of retinal artery 1	
Cyanosis at birth	5				
Vomiting, fever, scoliosis	1				

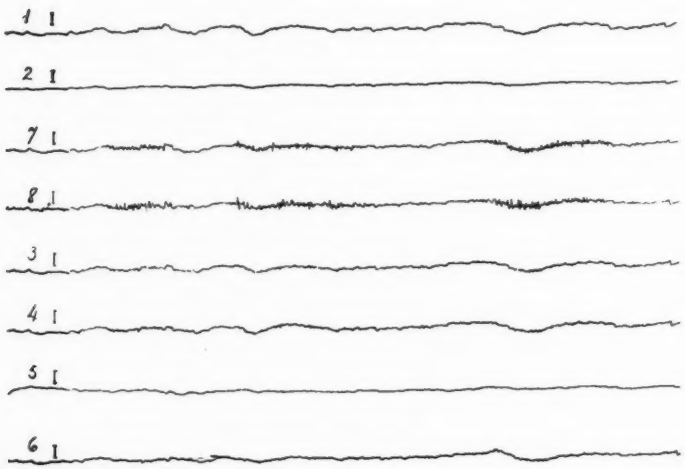


FIG. 3. Electroencephalographic changes in a case of anencephaly, showing microvoltage with muscle artefact in temporal leads.

with microphthalmus, in another, with abnormal distribution of the retinal artery.

The incidence of ophthalmic impairment in hydroencephalodysplasia is shown in Table 1.

ELECTROENCEPHALOGRAPHY

In electroencephalographic studies of 7 patients there were 4 instances of microvoltage, almost iso-electric activity interrupted by frequent muscle

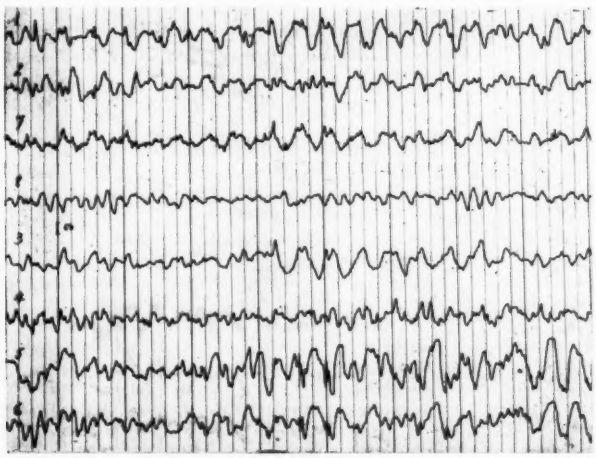


FIG. 4. General slow frequency with spike and wave activity in the occipital pole in a patient with bilateral frontoparietal porencephaly.

potential artefacts in the temporal leads (Fig. 3) or electrocardiogram transmissions. In the remaining 3 there were larger amounts of brain, and in these low voltage spikes were localized or scattered throughout the hemisphere superimposed in a slow 2–3 per second generalized activity (Fig. 4).

RADIOLOGICAL STUDIES

Simple roentgenograms of the skull revealed the enlarged head, bulging fontanels and other findings similar to those seen in congenital hydrocephalus. In one patient the head was small with premature closure of the sutures, and on the skull were digital-like impressions which suggested a certain degree of intracranial pressure. We have called these impressions pseudoconvolutional markings since there is no convolutional pressure and the resulting impressions are abnormal. Usually these are fewer in number and larger than true convolutional markings, and probably are equivalent to the local thinning of the skull so often found overlying a porencephalic cyst.

FIG. 5. Ventriculogram showing a malformed brain.

Often pneumoencephalography is contraindicated but ventriculography reveals a typical picture. Usually the malformation is gross (Fig. 5), and if the ventriculogram is performed with the injection of a rather large amount of air, the roentgenographic images silhouette the anatomical facts as shown in Fig. 12. Probably the two most constant findings are: (i) the absence of some amount of cortex, varying from a small portion, porencephaly, to the entire cortex, apallium; and (ii) the brain stem and surrounding malformed structures vary in amount and are seen as a triangular shadow in the base of the sagittal plane. Air studies are not well tolerated if the malformations are large, and not infrequently are followed by respiratory syncope or infection.

Probably the angiographic studies present the most pathognomonic picture (Figs. 6 and 7). The internal carotid is hypoplastic in the neck, thus making operative procedure difficult. In extreme cases of anencephaly all the important cerebral blood vessels may be absent and the internal carotid artery may end in a small bunch of fine blood vessels. In less severe defects as porencephaly the collateral branches from the middle cerebral artery may be reduced in number and localized in certain areas of the skull. The Sylvian vessels may be devoid of collateral branches ascending to the cortex or even without any collateral branches.[11,21,24] Care should be taken not to mistake the superficial temporal or the meningeal arteries for the cerebral vessels.

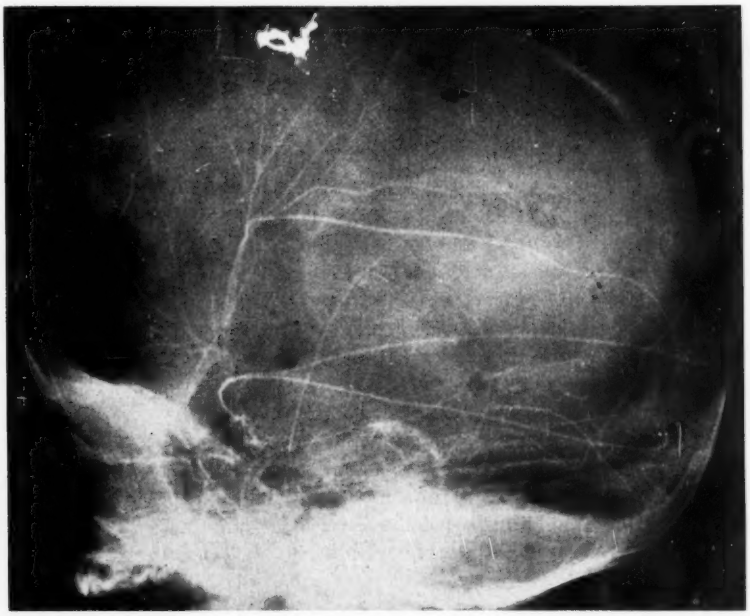


FIG. 6. Angiogram of same case as in Fig. 5, showing middle cerebral artery without collaterals.

NEUROLOGICAL SYMPTOMS

A certain degree of psychomotor arrest was present in all patients and evident in the "indifferent facies" previously mentioned. Motor symptoms such as rigidity, hemiplegia, generalized hyperreflexia and clonus were not uncommon. During the first few months of life a baby's behavior is limited to the most primitive reactions, and not infrequently a certain degree of undevelopment is not recognized by the mother or physician. However, behavior tests made after the fourth week are very nearly accurate and will indicate a certain degree of immaturity.

Convulsions, either generalized or Jacksonian, were noted in half the patients. These occurred during the first few days of life but recurred frequently thereafter.

Punctures of the fontanels and spine were made in the majority of the cases. The cerebrospinal fluid pressure varied in amount from 18 to 45 cm. of water in the ventricle, with the patient horizontal and asleep. Occasionally no fluid or only a few drops of a very low-tension cerebrospinal fluid was obtained. The fluid was clear, transparent and without changes except for one specimen in which the albumin was 44 mg. per cent. Specific tests for lues were negative.

Dandy's phenolsulfonphthalein test on 13 patients revealed 8 with a communicating disorder and 5 with a noncommunicating type.

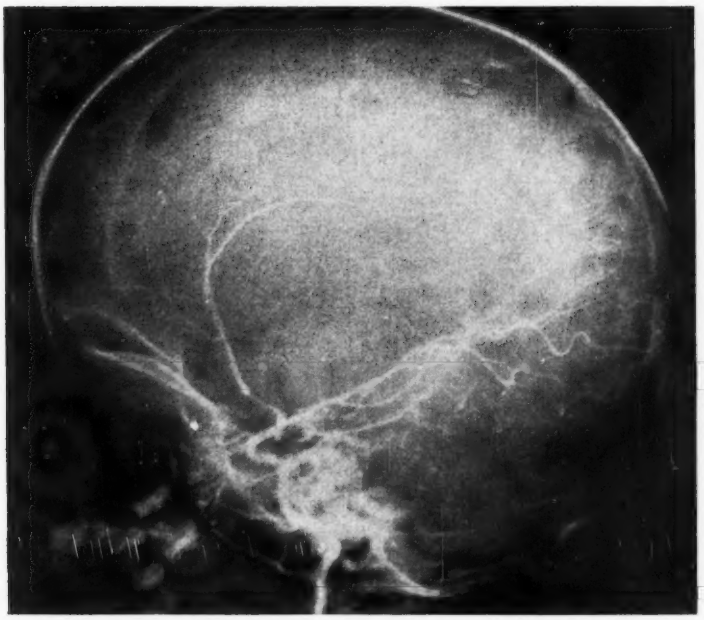


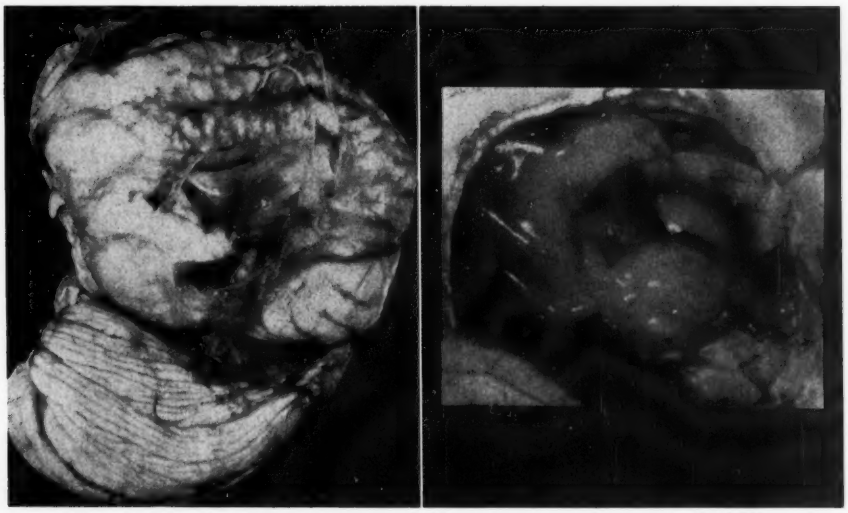
FIG. 7. Angiogram showing middle cerebral artery without collateral branches to the frontoparietal region. The anterior half of the right hemisphere was missing in this child.

PATHOLOGICAL FINDINGS

The pathological data were collected from surgical studies which included 10 exploratory craniotomies, and 3 from postmortem verifications.

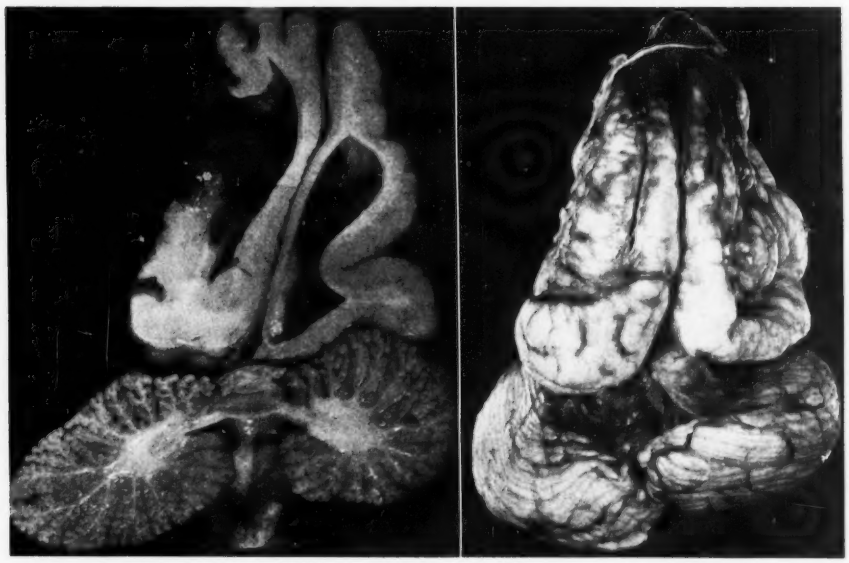
The findings from the autopsies are as follows: a well developed meningeal membrane; a dura mater irrigated by normal blood vessels, and with a well formed falx, tentorium and other normal structures; and arachnoid attached to the brain. Usually in the area with no cortex the arachnoid remained close to the dura mater and a large amount of cerebrospinal fluid was present. In 2 instances the arachnoid remained next to the malformed brain, and here continuity of the arachnoid covering the defect was lost. Cerebrospinal fluid had collected subdurally, so-called external hydrocephaly (Fig. 8).

Grossly the diencephalon in all patients was well developed. The thalamus and striatum crossed by an apparently normal choroid plexus appeared as two rounded masses in the base of the skull (Fig. 9). In the 3 autopsied cases the ventricular systems including the aqueduct were completely developed (Fig. 10). The defect was largely in the cortex, and here, more frequently the parietomotor area was missing. In the remaining cortex there was no flattening of the convolutions as in cases of increased intracranial pressure but convolutional abnormalities such as microgyri or extensive



FIGS. 8 and 9. (*Left*) An example of so-called "external hydrocephalus." Malformed cerebral hemisphere showing a large porencephalic cavity and tearing of the arachnoid, allowing the cerebrospinal fluid to escape to the subdural space.

(*Right*) Operative view of cranial contents in a case in which the right cerebral hemisphere was missing. The bilateral rounded masses are the diencephalon. The remaining nerve tissue is cortex of the occipital lobe, and mesial aspect of the hemisphere. The choroid plexus is well developed.



FIGS. 10 and 11. Autopsy findings in a case of anencephaly. (*Left*) Coronal section of the brain. Note abnormal formation of the gray matter and well developed ventricles. (*Right*) Occipital view showing malformed microgyri and areas of lissencephaly.

areas of lissencephaly were found (Fig. 11). The arteries had very few or no collateral branches (Figs. 6 and 7) and veins were noted jumping from the cortex to the longitudinal sinus in the midline.

Microscopically the dura mater appeared normal. The arachnoid was extremely fibrous and in many places the subarachnoid space was undeveloped or obliterated. The cortex also appeared essentially normal in the best areas seen grossly, but zones of microgyri and lissencephaly indicated an extremely abnormal cytological organization. Sometimes the fibrous tissue from the arachnoid had broken through the pia mater and grown into the cortex. The fibrous glia were more numerous in the zones of microgyri, and oligodendroglia were more numerous in the white matter. In no area was there any evidence of inflammatory reaction.

DISCUSSION

Developmental errors of the nervous system are not infrequently found associated with intracranial pressure. In a series of 135 consecutive cases of hydrocephalus in infants, we have demonstrated largely by surgical methods that over 50 per cent show some type of malformation of the nervous system. The percentage may be higher if only the congenital type is considered. Our findings and others described in the literature are listed in Table 2.

Malformations numbered 1 through 7 are located largely in the cerebrospinal fluid pathways so as to interrupt circulation in some way, and probably act as the cause of hydrocephaly. In these cases the main disturbance is in the pathway and the consequent hydrocephalus would gradually

TABLE 2

*Verified malformations of the nervous system associated with increased intracranial pressure, cerebrospinal fluid hypertension**

1. Septum of the foramen of Monro
2. Cyst of the septum lucidum or cavum of Verga
3. Maldevelopment of the aqueduct of Sylvius
   a. Stenosis
   b. Atresia (forking)
   c. Septum
4. Atresia of the foramen of Luschka and Magendie
5. Dysraphy
   a. Rachischisis (Arnold-Chiari)
   b. Cranioschisis (encephalocele)
6. Lissencephaly†
7. Closure of the subarachnoid space
8. Vascular and convolutional malformation; isolation of the choroid plexus in masses
9. Absence of corpus callosum; absence of vermis; fusion of the cerebral hemispheres
10. Porencephaly
11. Dysplasias
12. Anencephaly (hydroanencephaly)

* Symptoms of increased intracranial pressure in tuberous sclerosis and von Recklinghausen's disease are usually caused by obstruction in the cerebrospinal fluid pathways by a tumor or tubercle.

† Not seen by the authors. Described in literature.

destroy the brain. Patients in this category deserve prompt surgical attention since the earlier the condition is recognized and treated the better the ultimate prognosis if hydrocephaly can be arrested. Malformations numbered 8 are usually defects with little or no pathogenesis of increased cerebrospinal fluid pressure. Those malformations numbered 9, 10, 11 and 12 are considered characteristic of hydroencephalodysplasia since the main defect is in the brain itself rather than in the cerebrospinal fluid pathways.

Edinger,[10] who apparently was the first author to call attention to the possible relationship between porencephaly and anencephaly, considered the latter an extreme form of porencephaly. This opinion has been held by others. In Fig. 12 we have shown diagrammatically the anatomical findings from our observations in surgery. The amount of brain varies but we believe transition can be traced from simple porencephaly on one extreme to anencephaly on the other.

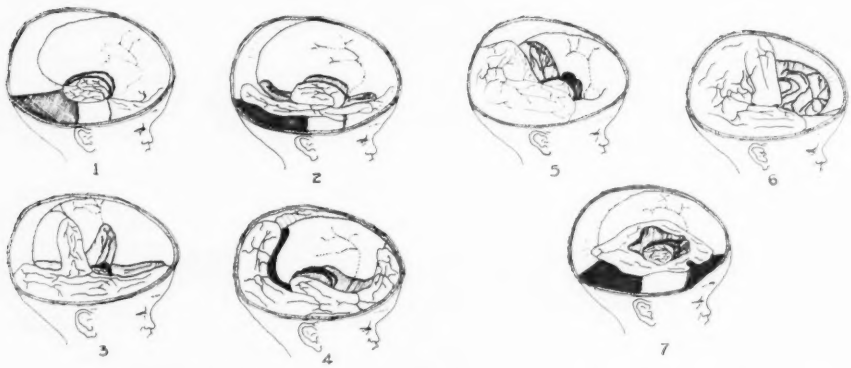


FIG. 12. Diagrammatic composition of the malformations found in hydroencephalodysplasia, ranging from hydranencephaly (1) to porencephaly (6) and external hydrocephaly (7).

After studying these malformations we have concluded that abnormalities previously described as anencephaly, congenital porencephaly,[1]* and other extensive defects or dysplasias are related and should be considered as a separate group since they have anatomicoclinical similarities and probably the same cause and related pathogenesis of pressure. Although the pathological findings in a number of the cases here reported have been largely surgically diagnosed and without a complete postmortem study, we have concluded that the brain malformations grossly seen are similar to those found in the necropsy of monsters, internal and external hydrocephalus, microcephalus, oligophrenia, epilepsy and others under the heading of

* We should differentiate between congenital porencephaly, in which condition the cause acts before birth, and acquired porencephaly, the term accepted by many authors to describe the loss of tissue that may follow the action of any pathological condition of the brain.

chronic encephalopathies. Therefore *hydroencephalodysplasia* would not be considered a different malformation in itself but an expression of a disturbance of the intracranial fluid balance in patients harboring gross dysplasias of the brain. In many of these dysplasias increased cerebrospinal fluid pressure would be evident in utero but in others symptoms of hypertension would appear in the first few years of life or even later.

Several instances have been reported of the development at varying times in life of increased cerebrospinal fluid pressure in patients with congenital porencephalic cysts. Love and Groff[16] reported an operation on a 9-year-old boy with symptoms of intracranial pressure. A huge parieto-occipital porencephalic cyst was found which drained into the lateral ventricle. Recovery following the operation was complete. Recently Drew and Grant,[9] in a review of benign cysts of the brain, reported 3 cases of what was probably porencephaly and in which there were definite symptoms of increased intracranial pressure. In 2 patients the cyst was not in communication with the ventricle but it was in the other. One patient died but the remaining 2 were asymptomatic 6 or 7 years after the opening of the cyst in the ventricle and removal of the walls. Two patients in our series harbored a huge porencephalic cyst. Chronic increased intracranial pressure was evidenced by enlargement of the head, positive Macewen's sign and opening of the sutures at the age of 2 years.

Surprisingly, the larger the defect in the brain the more frequent is the evidence of increased intracranial pressure. Hydranencephaly was recognized and described more than a century ago but reports of minor defects such as porencephaly are rather scarce.

In cases in which no symptoms of pressure are evident at birth the anomalies are usually associated with some degree of microcephaly and a syndrome of convulsions, neurological defects and mental derangement of varying degrees. After microcephaly has been established, any increase in pressure may be demonstrated by the pseudoconvolutional markings seen in the roentgenograms. These markings are larger in size and fewer in number than the true convolutional markings seen in craniostenosis; however, a differential diagnosis may be difficult. The pseudoconvolutional markings are probably the result of pressure of the bulging arachnoid covering of the cyst and restriction by the blood vessels that cross this membrane. Another modality of these changes is seen in certain cases of porencephaly in which a localized thinning of the skull reveals the location of the cyst. This is particularly true in the later stages.

In infants the appearance of increased cerebrospinal fluid pressure is rapidly shown by a noticeable enlargement of the head closely resembling that seen in internal hydrocephalus. This condition may be so diagnosed unless a careful clinical evaluation indicates the characteristic symptomatology, and complementary studies confirm the diagnosis. The main points of differentiation between congenital hydrocephalus in infants and hydroencephalodysplasia are shown in Table 3.

TABLE 3

Differences between hydroencephalodysplasia and congenital hydrocephalus

Hydroencephalodysplasia	Hydrocephalus
Clinical	
Expressionless face	Intelligent expression of face
Enlarged and translucent head	Enlarged head
Optic atrophy and ophthalmoplegia	Absence of neurological findings unless hydrocephalus quite advanced
Paralysis and spasticity	
Psychomotor undevelopment	
Convulsions	
Radiological	
Typical pneumoencephalogram and angiogram	Typical pneumoencephalogram and angiogram
Electroencephalographic	
Absence of electrical activity	Asymmetry of sleep spindles
Asymmetry of voltage	Slow rhythm
Focus	Focus, frontal or occipital
Puncture	
Large amounts of fluid, subdural	Negative subdural puncture
Pathological	
Agenesis of large portions of the encephalon	Nearly normal encephalon
	Defect largely in the cerebrospinal fluid pathways
Mechanism of Pressure	
Imbalance in production-absorption of fluid	Obstruction of cerebrospinal fluid pathways
Genesis	
Exogenous factor acting late in embryogenesis	Genetic factor acting very early in embryogenesis
Prognosis and Evolution	
Tendency to stabilization of pressure	Progression of hydrocephalus
Treatment	
Puncture or, in some cases, evacuation	Careful surgical management

A clinical differentiation may be impossible without radiological assistance in the following two conditions: (i) advanced internal hydrocephalus with destruction of the brain; and (ii) before the second month of age since the immaturity of the nervous system does not always reveal a quite noticeable symptomatology.

The increased pressure may not be important and in this event the patient appears as a chronic encephalopathic with bulging fontanels and a somewhat enlarged head. Eventually the pressure may stabilize, or in rare instances the equilibrium may break down later in life after the sutures of the skull have closed, with resulting symptoms of acute cerebrospinal fluid pressure. Examples of the latter condition are those reported by Love and Groff,[16] Drew and Grant[9] and others.

One of the most interesting facts about this condition is the tendency of

the symptoms of pressure to stabilize if evacuation of the cyst is carried out, although in some instances we have seen recurrence of pressure and progression to death.

From the pathological findings this disorder appears to be largely a defect in the telencephalon, more often in the neopallium. Usually, even in the more extreme conditions, a certain amount of cortical tissue is present at the base of skull around the midbrain or on the midline, occipital or frontal, and produces the characteristic appearance of a triangle in the roentgenograms. From a practical standpoint a newborn baby is functionally decorticated, consequently the behavior of an abnormal newborn baby may be very similar to that of a normal baby. Only later when functional integration of the cortex should appear can symptoms of immaturity or definite neurological defect be discovered. The expressionless or "indifferent facies" and the ophthalmological changes so characteristic of this condition and so important in differentiation of this condition from internal hydrocephalus are not noticeable before the third or fourth month of life.

Usually convulsions, another frequent symptom of these patients, do not occur in congenital hydrocephalus, and their appearance in a suspected case should direct attention to hydroencephalodysplasia, if birth hemorrhage and meningitis can be ruled out.

Patients in the early stages of hydroencephalodysplasia appear to be poor surgical risks. Two of our patients suffered severe respiratory failure following ventricular puncture. Barbiturates are not well tolerated as evidenced by frequent respiratory depression. Four patients died, 2 following ventriculography and 2 in shock following exploratory craniotomies. Two others died of respiratory disorders about a year after admission to the hospital. Severe developmental arrest, convulsions, spasticity and amblyopia were evident in varying degrees in 4 patients who were traced from 1 to 4 years. The only patient in a better condition was a child who had a minor type of dysplasia and was able to walk and say a few words at the age of 4 years.

The mechanism of production of increased pressure of cerebrospinal fluid differs somewhat in hydroencephalodysplasia from that in congenital hydrocephalus. Dandy's phenolsulfonphthalein test demonstrates either a communicating or noncommunicating type of hydrocephalus. The possibility of obstruction of the cerebrospinal fluid pathways alone as the cause of pressure is difficult to establish since in all cases there was a large communication between the cortical subarachnoid space and the ventricle through the area in which the brain was missing. The increase in cerebrospinal fluid could be either a transitory or a permanent result of the imbalance between the production of fluid by the choroid plexus and decreased absorption caused by the absence of an extensive area of the brain and partial closure of the subarachnoid space, lissencephaly, with consequent decrease of surface for absorption of fluid. In agreement with this theory is the fact that in a number of hydroencephalodysplasias an increase in cerebrospinal fluid pressure has been arrested by evacuation of the cyst surgically or simply by tapping

the cyst. In some cases the symptoms of fluid imbalance do not appear early in life. One explanation is based on inadequate absorption of fluid, another, mentioned by Drew and Grant,[9] on the presence of a yet unknown factor.

It is difficult to understand the development of such extensive brain malformations in the early stage of embryogenesis without evidence at the same time of some defect of the mesoderm which is so closely related at that period. Spatz[22] has shown that the brains of newborn animals liquefy and disappear on interruption of the blood supply, leaving only cysts.* Crosby's[3] opinion from the study of 2 cases of hydranencephaly was that the development of the brain was normal to at least the fifth month of prenatal development but thereafter underwent regression. Substantiating this theory is the fact that in all of our patients the skull and meningeal membranes were rather normal in appearance. In 1 patient the entire anterior halves of the hemispheres were missing. The mother had attempted abortion in the fourth month of pregnancy by the injection of formaldehyde into the uterus. Three patients had histories of recurrent bleeding from the sixth to the eight month of pregnancy. The mother of another patient was poorly nourished and had suffered a severe abdominal trauma at the seventh month of pregnancy. In 7 patients some type of brain disorder was indicated immediately after birth by cyanosis, convulsions, difficulty in feeding, hyperirritability or continuous crying. We believe these disturbances are not the cause of hydroencephalodysplasia but the result of the inability of the dysplastic brain to adapt the individual to the environment.

We repeatedly questioned the parents of the patients and found in only the 5 cases mentioned above, any etiological factors such as trauma, fever or intercurrent diseases that might have accounted for the maldevelopment. The fact should be noted that the cases reported in this study are largely from a charity hospital, and many of the patients are peasants whose diet is unbalanced, poor in meat and vitamins and rich in starch. A genetic factor was a possibility in only 1 patient. A hydrocephalic cousin had been successfully treated 4 years before this report for occlusion of the aqueduct, and a distant relative, a hydrocephalic, died in the first few months of life.

SUMMARY

Under the term *hydroencephalodysplasia* the authors have discussed a group of cases which are characterized by extensive dysplasia of the brain and increased cerebrospinal fluid pressure. The defect is in the brain itself rather than in the cerebrospinal fluid pathways, thus differentiating hydroencephalodysplasia from congenital hydrocephalus.

Clinical points include enlargement of the head in varying proportions, psychomotor arrest, convulsions, and neurological and ophthalmological

* After this paper was finished the work of F. S. Vogel and J. L. McClenahan came to our attention (Anomalies of major cerebral arteries associated with congenital malformations of the brain, with special reference to the pathogenesis of anencephaly. *Amer. J. Path.*, 1952, *28:* 701–723). They discussed arterial anomalies in 14 anencephalic monsters. Furthermore, they were able to reproduce this malformation by occluding the internal carotid artery in the chicken embryo.

defects. Ventriculograms and angiograms are characteristic. Electroencephalographic findings differ from those of internal hydrocephalus.

An increase in cerebrospinal fluid pressure may appear in the uterus but more frequently appears during the first 2 years of life, or even later.

Pathology reveals all degrees of agenesis of the telencephalon from porencephaly to anencephaly, and includes cyto-architectonic changes and thickening of the arachnoid membrane with closure of the subarachnoid space in certain areas.

The malformation seems to be related to an acquired injury occurring late in embryogenesis.

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THE USE OF ORLON FOR DURAL REPLACEMENT

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THE continuity of the dura mater is a prime necessity. Various processes may interrupt this continuity, such as a tumor growing in its inner surface, a fracture of the skull associated with a gross laceration of the dura mater, and certain surgical procedures that may destroy part of this membrane.

The herniation of the brain and the leptomeninges through the dural defect may have undesirable consequences. Sometimes meningocerebral adhesions are formed; even craniocerebral erosions may occur.[6] If the arachnoid breaks and there is an underlying fracture of the cribriform plate or of the petrous portion of the temporal bone, the cerebrospinal fluid will leak, and rhinorrhea or otorrhea may result. These fistulas connecting with the pneumatic cavities of the face may give passage of the air into the skull, and pathogenic agents may spread into the cranial cavity causing infections such as meningitis or brain abscesses.

As the dura mater does not regenerate with ease,[5] any of the above complications may occur with undesirable sequelae. Therefore, the closure of a dural defect is of paramount importance in neurosurgery. In the past many methods have been tried:

(a) Grafting.[1,2]

(b) Use of albumin as a base substance for the regeneration of the dura mater: products prepared from human fibrinogen and human thrombin;[8] fibrin foams and films—Gelfoam-fibrin.

(c) Prostheses. Replacements of the dura mater have been attempted in different ways[3] using metal foils, polyethylene, celluloid, cellophane, rubber tissue, etc.[7]

All these methods have brought a great advance in the treatment of dural defects, but each method can be seriously criticized. The grafts may deteriorate. Fibrinogen and thrombin sometimes encourage the formation of septic foci. Metal foils are too rigid to take over the functions of a flexible dura mater. Most prosthetic appliances thus far have acted as a foreign body producing a tremendous reaction with formation of a membrane or an increase of the scar tissue.

The ideal material would be one of nonbiotic origin which would have the advantages of being readily available in all sizes, without the necessity of meticulous processing, which would protect the brain without producing reaction of the glial tissue or meningocerebral adhesions, and which would keep the normal shape of the hemispheres and spinal cord.

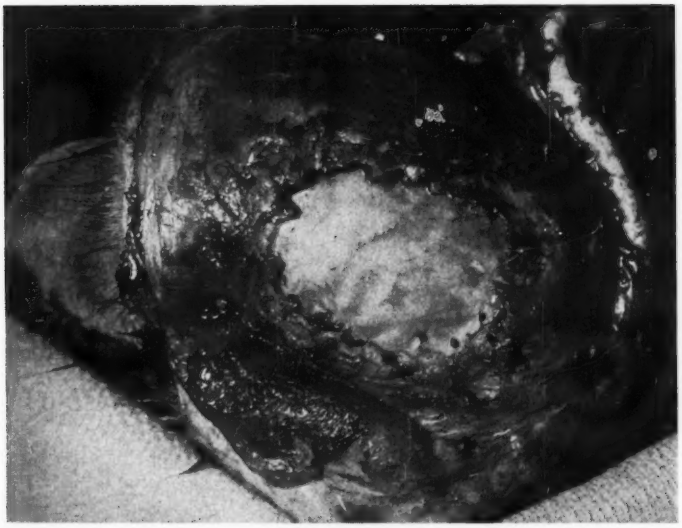

FIG. 1. Orlon prosthesis after removal of the bony calvarium. The dura mater has proliferated over the edges of the Orlon and sealed them.

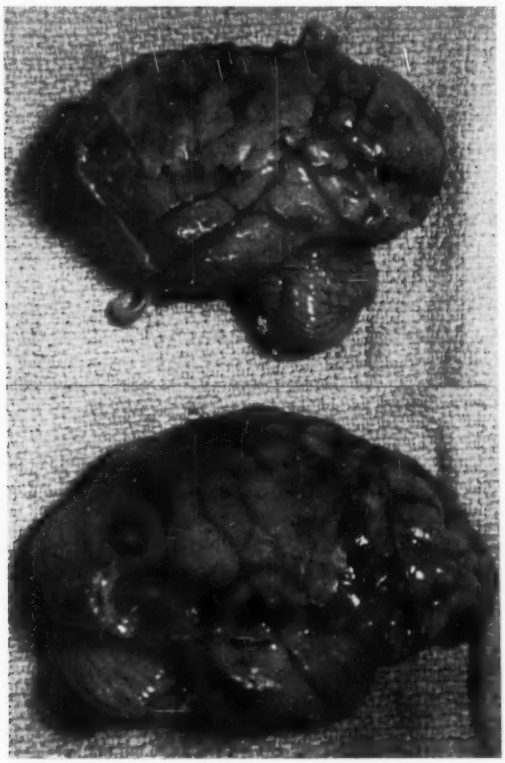

FIG. 2. Extent of dural replacement in 2 of the monkeys

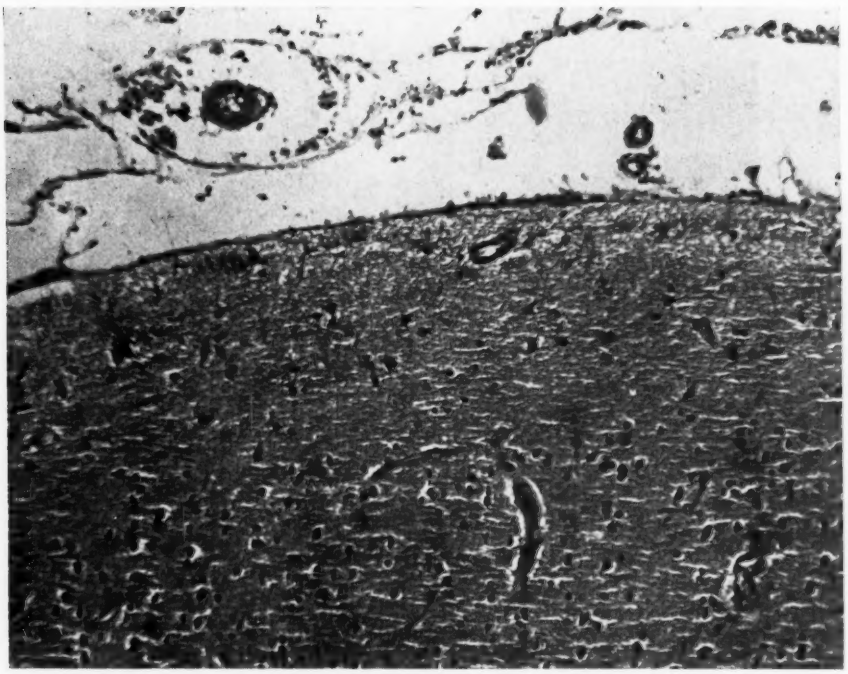

FIG. 3. Photomicrograph of the brain beneath the Orlon after 15 months. Note the intact pia-arachnoid and absence of cellular reaction. This area is over the orbital plate.

The qualities of this ideal material should be as follows: (i) It should resemble the dura mater in flexibility, thickness and strength. (ii) It should be biologically inert. (iii) Its internal surface must be extremely smooth so it will not harm the delicate surface of the brain. (iv) It must be waterproof.

The idea of using a cloth-type prosthesis for dura mater came to us by observing the experimental work on prosthesis of arteries.[4] The best known cloth-type plastic materials are Vinyon, Nylon, Orlon, and Dacron.

MATERIAL AND METHODS

Vinyon leaks excessively; therefore, it is undesirable for our purposes. It has been observed that Nylon precipitates the proteins and thus causes an ever-thickening membrane when embedded in living tissue. Dacron and Orlon are two materials that seem to offer good possibilities for prosthesis. As strands of Orlon are very uniform and smooth and produce a minimum reaction of tissue, we chose this material for our experiments, using 75 to 100 weave of type 81 Orlon fiber (DuPont).

It is always desirable to wash the cloth thoroughly because as the fibers are processed, a certain amount of oil and foreign matter may accumulate on the surface of the fibers. The material is autoclaved.

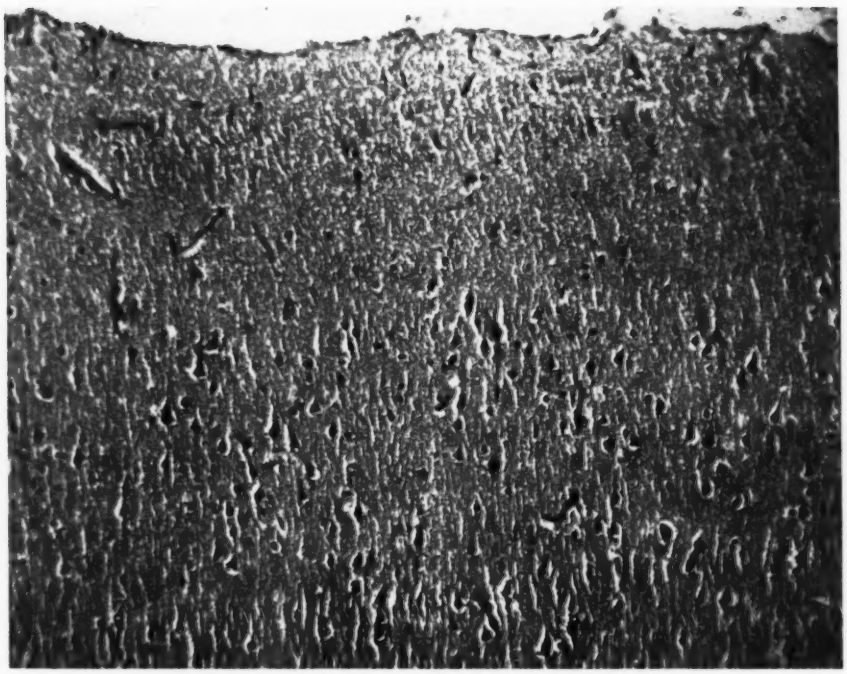

FIG. 4. Photomicrograph showing another section from same area as in Fig. 3.

Seven adult *Macaca mulatta* monkeys were used in our experiments.

Group 1. In 5 monkeys under Nembutal anesthesia and sterile technique, a hemicraniotomy was performed exposing the dura mater. An oval dural defect measuring about 4 cm. was made. With a fine needle the brain was lacerated in 2 animals and a prosthesis of Orlon was placed over the defect. It was sewn by separate stitches of silk, and care was taken to fold the edges so that the fibers of the fabric would not rip apart. The bone was placed back into position with steel wire. The skin was sewn with separate stitches of braided Nylon.

Two of these monkeys were sacrificed a week later. No great difference was found in the prosthesis or the brain as compared with their appearance on the day of operation. Three monkeys were sacrificed 5 months later. The Orlon was loosely adherent to the bone to the same degree that the dura mater normally would be attached to the bone. Small adhesions were present (Fig. 1). Care was taken to remove the bone flap without injuring the preparation. The edges of the prosthesis were covered by tissue hiding the stitches completely. The rest of the skull was removed with rongeurs. Then the dura mater was sectioned around the Orlon (Fig. 2) in order to observe it *in situ*. The inner surface was completely free. The brain appeared healthy and on microscopic examination it showed no reaction whatsoever. There was no attempt to form an envelope around the prosthesis. The neighboring tissues did not show any foreign body reaction.

Group 2. In the other 2 monkeys a frontal craniotomy was performed. The frontal lobe was lifted, and the cribriform plate was broken. As there was not enough space to sew the prosthesis about the dura mater, it was fixed by a few separate stitches.

These monkeys were under observation for 15 months, and no abnormality was seen. Particular attention was paid to any nasal discharge. The cerebrospinal fluid was checked at 8 months in 1, and it was normal in appearance, and contained no cells. At autopsy the general appearance of the graft did not vary much from the appearance of the grafts previously described. The surface of the brain appeared clean and neat (Figs. 3 and 4). However, the material was firmly attached to the bone, and it was impossible to scrape it out without causing further damage.

Both groups of our animals were alert and active. No neurological deficit was observed in any of them. Electroencephalograms were normal.

On the basis of these experimental findings a dural prosthesis and repair were performed on a 37-year-old woman who had a frontal abscess. She has been followed for 3 months. No abnormality has been noticed thus far.[9]

SUMMARY

1. Orlon seems to be an ideal fabric for prosthesis of the dura mater. It is readily available, easy to handle and to store, it is strong and waterproof, and produces a minimum reaction.

2. This material has been tried on 7 monkeys. Two of them were observed for 15 months, and no abnormality, neither clinically nor pathologically, was observed.

3. One 37-year-old woman with a brain abscess had an Orlon dural prosthesis. No abnormality has been observed after 3 months.

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CORTISONE AS AN AID IN THE SURGICAL TREATMENT OF CRANIOPHARYNGIOMAS*

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THE surgical treatment of the craniopharyngiomas has been attended by a high mortality rate in the past, especially when total removal was attempted. An operative mortality of 41 per cent, and even higher, has been reported.[1,2,4,5,8] The close relationship of most craniopharyngiomas to the hypothalamus exposes the latter to operative trauma and in the past, postoperative death has usually been attributed to this cause. Another factor that was thought to contribute to this high mortality, and with good reason we now believe, was the longstanding direct pressure effect of the tumor on the pituitary gland itself.

Patients in whom the anterior lobe of the pituitary gland has been compressed with resultant hypopituitarism are a poor risk for any type of surgical procedure. They may die of adrenocortical collapse either during or shortly after operation. Even in patients without preoperative evidence of hypopituitarism, removal of a craniopharyngioma may necessitate extension of the dissection into the sella turcica with resultant damage to the hypophysis. This may lead to loss of endogenous adrenocorticotrophic hormone upon which survival depends.

At the University Hospital the craniopharyngiomas have been approached for the past 30 years with the idea that total removal would be performed whenever possible. In cases in which the tumor extended beneath the optic chiasm to invade the third ventricle, or when it was densely adherent to the optic nerves or internal carotid arteries, only the accessible portions were removed. In the series reported by Gordy, Peet, and Kahn[4] total removal was believed to have been accomplished in 25 of 51 cases. Recurrence was known to have taken place, however, in 6 of these patients. The operative mortality in the entire series was 41 per cent. The majority of deaths were attributed to severe hypothalamic reactions as a result of operative trauma.

We are reporting a series of 21 patients all operated upon by one of us (E.A.K.) since 1936. Since there has been little change in operative technique over this period of time, it is our opinion that the greatest variable affecting the markedly improved postoperative survival has been the use of

* Presented at the meeting of the Harvey Cushing Society, Quebec, Canada, May 17, 1955.

TABLE 1

Postoperative results, craniopharyngiomas
Definite hypopituitarism
Cortisone not used

No.	Age Sex	Estimated Removal of Tumor	Maximum Rectal Temperature	Time and Cause of Death
1	23 M	Incomplete	97.6°	3 hrs. p o. Circulatory collapse
2	33 M	Incomplete	106°	1st day p.o. Circulatory collapse
3	31 F	Incomplete	105°	1st day p.o. Circulatory collapse
4	38 F	Complete	103°	1st day p.o. Circulatory collapse

cortisone. With this drug it has been possible to prevent postoperative adrenocortical failure and to effectively combat hyperthermia, which in itself may cause death. With the reduction in postoperative complications that the administration of cortisone has permitted, total extirpation of these tumors should be attempted in many more cases. In 1952 Ingraham, Matson and McLaurin[6] reported good results with the use of cortisone in operations upon 5 patients with craniopharyngiomas and with ACTH in another pa-

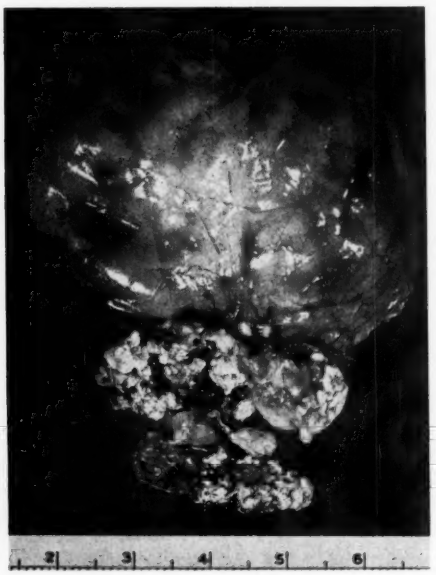

FIG. 1. Photograph of the specimen removed from Case 1 of the cortisone series, a 2-year-old child with signs of hypopituitarism preoperatively. Total removal was believed to have been accomplished. Only the irregular solid portion of the tumor contained calcium. Microscopically some pituitary gland substance was attached to the tumor. This child has done well postoperatively but blindness which was present preoperatively has persisted. (Reprinted with permission from Charles C Thomas, Publisher.[7])

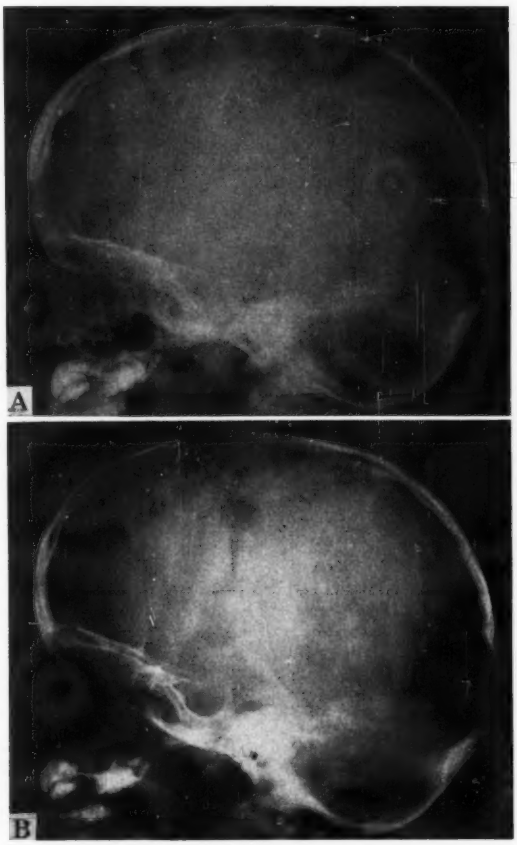

FIG. 2. (A) Preoperative roentgenogram of Case 6 of the cortisone series, a 4-year-old child with preoperative signs suggestive of hypopituitarism. Calcification can be seen within the sella turcica. (B) Postoperative roentgenograms taken 2 months later revealed complete absence of calcification in this region. The radiopaque substance seen in the upper portion of the sella is felt to be artifactual since it was not seen in other projections.

tient. These operations, however, were in general not as radical as those we have attempted.

Raaf, Stainsby and Larson[9] in 1954 reported on 18 consecutive patients with tumors in the region of the sella turcica who were prepared for operation with ACTH. There were no deaths in this series though in the only 2 patients with craniopharyngiomas total removal was not carried out.

CLINICAL DATA

Fourteen patients received no cortisone. Preoperatively at least 4 of these showed definite hypopituitarism and all died postoperatively. Table 1 shows the postoperative results in detail.

Ten of the 14 patients who received no cortisone did not show unequivocal signs of anterior pituitary deficiency prior to surgery. Table 2 shows the break-down in these cases.

The 7 patients who were given cortisone preoperatively had strikingly

TABLE 2

Postoperative results, craniopharyngiomas
Patients without unequivocal signs of hypopituitarism
Cortisone not used

No.	Age Sex	Date of Operation	Estimated Removal of Tumor	Maximum Rectal Temperature	Time and Cause of Death	Follow Up
1	3 F	1938	Complete	101°	—	Working as nurse.
2	29 F	1940	Incomplete	101°	—	Diabetes insipidus 5 mos. Died of recurrence, 2 yrs. 9 mos.
3	14 F	1940	Complete	108°	2nd day p.o. Circulatory collapse	—
4	3 F	1947	Complete	103°	—	Diabetes insipidus 5 yrs. Stormy convalescence; 1 episode of circulatory collapse. Alive and well.
5	2½ M	1947	Complete	104°	—	Alive and doing well.
6	4 F	1949	Complete	106.4°	1st day p.o. Circulatory collapse	—
7	5 F	1949	Incomplete	103.6°	1st day p.o. Circulatory collapse. Operated upon twice before	—
8	9 F	1951	Incomplete	104°	—	Diabetes insipidus 18 days. Recurred 17 mos. p.o. Alive.
9	22 M	1951	Complete (?)	104°	—	Died 1 yr. p.o. Given ACTH during and after surgery.
10	47 F	1952	Incomplete	102°	—	Drowsiness 4 mos. p.o. Died 9 mos. p.o.

uneventful postoperative courses. The maximum temperature was 103° rectally and this subsided within 24 hours (Table 3). One of these patients, however, died with hyperthermia 4 days after reoperation for recurrent tumor in spite of again receiving supplemental cortisone therapy. In addition, a Torkildsen procedure ultimately became necessary in another of these cases. Preoperative cortisone preparation was not carried out at this time and 38 hours after operation the patient suddenly collapsed into irreversible shock from which she never recovered. Death occurred on the 4th postoperative day.

TABLE 3

Postoperative results, craniopharyngiomas
Prepared with cortisone

No.	Age Sex	Date of Operation	Estimated Removal of Tumor	Maximum Rectal Temperature	Follow Up
1	2 M (probable hypopit.)	1951	Complete	101.6°	Diabetes insipidus 1 mo. Alive and well. Preoperative blindness continues.
2	17 M (hypopit.)	1951	Incomplete	102.8°	Diabetes insipidus 1 mo. Died 3 mos. later. 3 days p.o. recurrence in spite of preparation with cortisone.
3	2½ F (? hypopit.)	1951	Complete	102.4°	Diabetes insipidus persists. Alive and well.
4	4 F	1952	Incomplete	103°	Died 2 mos. later in circulatory collapse 4 days p.o. Torkildsen procedure for recurrence. Not prepared with cortisone at this time.
5	11 M	1954	Complete (?)	102°	Diabetes insipidus. Alive and well.
6	4 F (probable hypopit.)	1954	Complete	102.8	Diabetes insipidus. Alive and well.
7	13 M (hypopit.)	1955	Incomplete	101.6°	Diabetes insipidus. Alive.

In summary, all of 7 patients who were prepared for craniotomy with intramuscular cortisone, and who received it throughout the period of surgical stress, survived the initial operative procedure.

Of the 5 surviving patients in the series receiving cortisone, it is believed that total removal of the tumor was accomplished in 4. All of these patients require maintenance doses of oral thyroid extract. One patient has complete panhypopituitarism and requires small doses of oral cortisone daily. Only 1 patient in the total series of 21 cases had diabetes insipidus preoperatively. This persisted 5 years postoperatively and then disappeared. In 6 cases diabetes insipidus developed postoperatively requiring intramuscular pitressin tannate in oil every 2 to 12 days to control the polyuria.

ENDOCRINOLOGICAL ASPECTS

Extirpation of the anterior lobe of the normally functioning pituitary gland abruptly interrupts secretion of four important hormones, each of which stimulates its specific "target organ": (1) somatotrophic or growth hormone, (2) the gonadotrophic hormones (follicle-stimulating hormone,

luteinizing hormone and luteotrophic hormone), (3) thyrotrophic hormone, and (4) adrenocorticotrophic hormone. Lack of trophic hormone secretion is signaled clinically by subnormal function of the target organ, and if all trophic spheres are involved a state of panhypopituitarism exists. It should be noted that one cannot assay the presence or absence of growth hormone in an adult, just as one cannot determine clinically the status of the gonadotrophic sphere in a prepubertal child. In addition, evaluation of adrenocortical status is frequently inconclusive in children because of practical difficulties in obtaining accurate urine collections and because urinary steroid

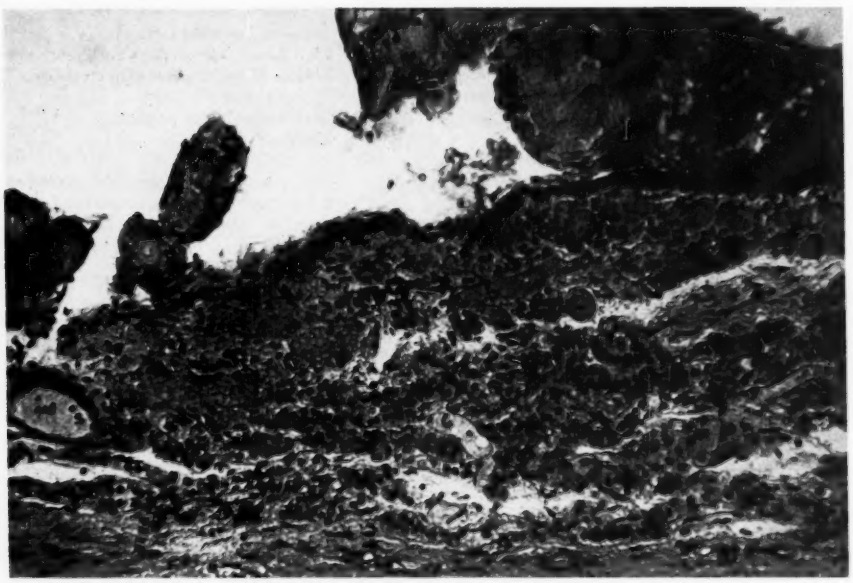

FIG. 3. Photomicrograph of the specimen removed from Case 6 of the cortisone series. Columnar epithelium typical of craniopharyngioma can be seen in the mid-portion of the field; below this, glandular structure characteristic of normal pituitary gland is present. It was thought, therefore, that the pituitary gland was at least damaged during the removal of the tumor and evidently partially removed.

values are low even in normal children. It should also be emphasized that the eosinophil response to a test dose of 25 mg. of intramuscular aqueous ACTH is not a reliable criterion of adrenocortical reserve, since this minimal stimulus may cause an entirely normal eosinopenia even though there actually exists dangerous incapacity of the cortex to respond to the major stress of surgery.

Of practical importance is the fact that, when decrease in anterior lobe function is gradual rather than abrupt—which may occur if a neoplasm either directly invades the adenohypophysis or causes atrophy by external compression—evidence of target organ deficiency usually appears in a selec-

tive and teleological sequence. *The trophic hormones least necessary for survival disappear first, while the one most vital for survival is the last to go.* Thus, growth hormone and gonadotrophic hormones disappear first, thyrotrophic hormone next and adrenocorticotrophic hormone last. In patients with craniopharyngiomas, therefore, one frequently finds delayed somatic growth, arrested sexual maturation, and manifestations of hypothyroidism, but with apparently intact adrenocortical function and reserve. On the other hand, if there is evidence of adrenal cortical insufficiency there are usually deficiencies in all other trophic spheres as well.

However, even if all preoperative data indicate entirely normal function of the adenohypophysis, one should not depend upon the continued integrity of the pituitary-adrenal axis in the precarious postoperative period. Preparation with adrenal steroids of patients with suprasellar lesions is still advisable for two reasons:

(1) Unless time-consuming tests of adrenocortical reserve are done, one cannot be sure that an apparently intact pituitary-adrenal system will be capable of responding normally to the stress imposed by surgery.

(2) In order to achieve gross total removal of the neoplasm it may be necessary to damage the adenohypophysis. Adrenocorticotrophic secretion could cease as of that moment and adrenocortical collapse might occur within 24 hours.

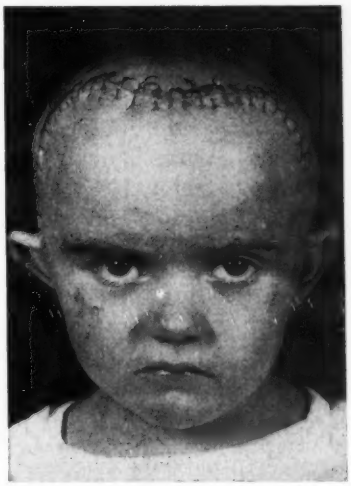

FIG. 4. Photograph of patient in Case 6 of the cortisone series, 48 hours postoperatively.

No complications from the cortisone itself have been observed before, during or after surgery. Because of the sodium- and water-retaining properties of large doses of 11,17-oxysteroids, possible aggravation of postoperative cerebral edema was originally accepted as a calculated risk. However, for the past 2 years the same program of cortisone administration has been used at this hospital in virtually all patients undergoing surgery in the pituitary area, and in only 1 instance has the postoperative course been as turbulent as in patients who were not prepared in this manner.

As a matter of fact, it has been consistently noted that the postoperative course in the cortisone-treated patients is impressively benign. Particularly striking is the almost complete absence of the hyperthermic crises which used to routinely follow surgery in the hypothalamic area, and which were attributed to post-traumatic edema and softening in this region. In the present series, the death of 5 of the 13 patients who did not receive cortisone was attributed, at least in part, to hyperpyrexia (rectal temperatures ranged

from 105° to 108°), whereas in the cortisone-treated patients at primary operation, the highest postoperative rectal temperature was 103°. A reasonable explanation for this property of adrenal steroids in preventing postoperative cerebral edema stems from the fact that brain tissue is predominantly cellular. Elliott and Yrarrazaval[3] have shown *in vitro* that cortisone reduces hydration of brain tissue; and Streeten[10] demonstrated in studies on human erythrocytes that cortisone induces a loss of intracellular water at the same time that extracellular water is being retained. Thus, by actively preventing retention of intracellular fluid, cortisone may have a specific effect in reducing postoperative cerebral edema.

In the present series cortisone (compound E) was used as the agent of choice, although hydrocortisone (compound F) may ultimately replace cortisone when an intramuscular preparation becomes available. Direct administration of the active adrenocortical compound obviates the risk inherent in using ACTH to stimulate secretion from the patient's own adrenals. As noted by Raaf and coworkers[9] who used ACTH on their patients undergoing parasellar surgery, 3 to 5 days of ACTH administration may be required to restore the involuted adrenal cortices to normal responsiveness. Moreover, it is recognized that the response to intramuscularly administered ACTH, either the aqueous or the gel preparations, is not always predictable because of variation in the rate of absorption or local inactivation in the muscle.

Cortisone administered intramuscularly is absorbed much more slowly than when given orally. Following an intramuscular injection, an effective pharmacologic response is not obtained for more than 24 hours, but steroid activity then persists at least 4 days. Conversely, when cortisone is given orally, its clinical effects are evident 4 hours later but disappear within 12 hours.[11] In patients scheduled for surgery the oral route is neither feasible nor dependable. The intramuscular route is therefore used and the program is begun either 36 or 48 hours preoperatively to ensure the existence of maximal steroid activity at the time of operation. We prepare adolescent and adult patients for surgery by administering 200 mg. of cortisone intramuscularly daily for 2 days preoperatively and on the day of surgery. Postoperatively the dose is usually tapered to maintenance levels, or withdrawn entirely, within a week. In general, small children are given half this dosage. In the weeks or months following cessation of supportive cortisone therapy the patient should be watched carefully for evidences of adrenocortical deficiency. Adrenal insufficiency secondary to pituitary failure is more frequently manifested by symptoms of hypoglycemia than by those of electrolyte loss, whereas the converse is true in primary adrenal failure. This is presumably because of the dual absence in panhypopituitarism of the antiinsulin activities of growth hormone and of adrenal corticoids, while only the latter are lacking in Addison's disease. When they occur, symptoms of cortical deficiency usually respond readily to 15 or 20 mg. of oral cortisone in divided doses daily.

If secondary hypothyroidism develops postoperatively, replacement therapy consists of 100 to 130 mg. (1½ to 2 gr.) of desiccated thyroid daily. Hypogonadism in patients past the age of puberty is treated with cyclic oral estrogen (e.g., 1 mg. of stilbesterol daily the first 20 days of each month) or intramuscular depot-testosterone (200 mg. of testosterone cyclopentylpropionate twice a month). There is, at present, no effective replacement for growth hormone deficiency.

Diabetes insipidus of postoperative onset may be transient or permanent, depending upon the reversibility of the surgical trauma to the hypothalamus or the pituitary stalk. Treatment is symptomatic, with 0.5 to 1.0 cc. (5 pressor units per cc.) of pitressin tannate in oil being given intramuscularly as often as necessary to control polyuria. Transient diabetes insipidus usually subsides within 2 or 3 months, but may last a year or longer.

SUMMARY

A series of 21 patients with craniopharyngiomas is presented. Each case has been approached with the idea in mind of total excision of the tumor if it seemed possible from X-ray studies or during operative exposure. Total excision is certainly possible at times, but only a long follow up or autopsy can establish this fact.

Six of the 14 patients who were not given cortisone died within 48 hours of their craniotomy either from hyperthermia or peripheral vascular collapse; 1 patient had persistent hyperpyrexia and expired on the 10th postoperative day, a mortality rate of 50 per cent.

In contrast, all 7 patients who received cortisone recovered uneventfully from their initial craniotomy.

The use of intramuscular cortisone before and after surgery of the craniopharyngiomas reduces postoperative mortality by: (1) preventing postoperative adrenocortical collapse, and (2) preventing postoperative hyperthermic crises, perhaps by minimizing postoperative edema.

No complications attributable to adjunctive cortisone therapy have been observed to date.

CONCLUSIONS

Preoperative and postoperative administration of intramuscular cortisone is recommended in all patients being prepared for surgery of the craniopharyngiomas. The same program is likewise indicated in any patient undergoing surgery in the pituitary-hypothalamic area. Proper preoperative preparation of the patient should lead to many more attempts at total removal of craniopharyngiomas.

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THE PROBLEM OF SPONTANEOUS SUBARACHNOID HEMORRHAGE WITH PROVEN ANEURYSMS*

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THIS paper is based on 60 consecutive cases of spontaneous subarachnoid hemorrhage in normotensive individuals who survived their initial insult. All were in the surgery age-group. The follow-up period ranges from 1 to 5 years. In 38 cases aneurysms were demonstrated: 33 were saccular and 5 arteriovenous. Eight patients with saccular aneurysms refused surgery. On conservative therapy 7 are now dead as a result of recurrent hemorrhage from their aneurysm (Table 1). The remaining 30 aneurysms

TABLE 1

Saccular aneurysms—conservative treatment
8 cases—7 deaths

Location	Age	Recurrent Hemorrhage (Time Interval)	Result
Middle cerebral	15	3 weeks	Died
Anterior cerebral	18	4 weeks	Died
Anterior cerebral	30	8 weeks	Died
Supraclinoid	40	4 weeks	Died
Posterior communicating	43	3 weeks	Died
Middle cerebral	24	4 weeks	Died
Anterior cerebral	51	2 weeks	Died
Anterior cerebral	56	2 years	Alive to date

were operated upon by direct intracranial attack. There were 4 operative deaths in the 30 cases and 1 patient became hemiplegic. No hypothermia was used in this series. Hypotensives were used in most cases.

One cannot overemphasize the value of stereoscopic angiography with sufficient projections to give the detailed relationship of the aneurysm and its neck to the parent vessel. With such information one can safely clip with a minimum of probing and dissecting—manoeuvres that invite rupture (Fig. 1). Postoperative angiograms were done whenever doubt existed concerning the complete obliteration of a saccular aneurysm. Repeated postoperative angiograms are a definite risk and not indicated unless roentgeno-

* Material presented at the Royal College of Surgeons meeting in Winnipeg, April 6, 1955, and the Harvey Cushing Society meeting in Quebec, May 18, 1955.

FIG. 1. (*Left*) Anteroposterior view of carotid angiogram showing saccular aneurysm at junction of anterior cerebral and anterior communicating. The aneurysmal sac hangs down and appears to rest on the tuberculum in this view.

(*Right*) Postoperative angiogram indicating complete obliteration of the aneurysm by a single clip placed on its neck with the circulation preserved beyond to the anterior cerebral branches.

grams demonstrate a change in the position of the clip or opening of the clip. To date there has been no evidence of recurrent hemorrhage in any of the 26 patients surviving surgery.

In 3 of the 4 cases in which surgical fatality occurred, the patients were operated upon while in coma and 2 in addition had multiple aneurysms (Table 2). The other patient died apparently of inadequate anterior cerebral circulation. He regained consciousness and movement in all four extremities. His conversation was always spontaneous but not always appropriate. He became progressively spastic and drowsy, dying 44 days postoperatively. Autopsy revealed extensive softening in both frontal lobes. The anterior cerebral vessel not bearing the aneurysm was an extremely small one; the one bearing the aneurysm had been clipped proximal to the anterior com-

TABLE 2

Saccular aneurysms—surgical deaths

Location	Age	Condition	Survival
Anterior communicating	48	Good	44 days
Anterior communicating & middle cerebral	57	Coma	18 days
Anterior cerebral & middle cerebral	55	Coma	24 hours
Anterior cerebral	40	Coma	28 hours

municating artery. There was no evidence of recurrent hemorrhage.

The patient rendered hemiplegic had an infraclinoid carotid aneurysm. This case is of considerable interest from the point of view of collateral circulation. The first stage was done under local anaesthesia. Pre-operative Matas test and angiograms had indicated a satisfactory collateral across the anterior communicating artery. At surgery, however, each time the internal carotid was occluded in the neck the patient became instantly but transiently hemiplegic without visual disturbance. After this was repeated three times, the occlusion was held for several minutes without re-appearance of the hemiplegia. The artery was doubly ligated. The patient had no further weakness although it was felt her margin of reserve collateral was very slight. The deprived carotid in such a case must be supplied entirely across the anterior communicating plus the posterior communicating artery if it exists. The ophthalmic is still in the system but may be contributing blood to the carotid or draining blood from the carotid.

Two weeks later the carotid was clipped at the clinoid without incident. Such a manoeuvre traps only one vessel from the available carotid circulation, the ophthalmic artery. The patient was completely relieved of her persistent pain but was also completely hemiplegic without visual disturbance. There are other possible explanations for the hemiplegia but one must consider the collateral from the ophthalmic artery as of great significance. Various authors have mentioned its potential contribution to the cerebral circulation following carotid occlusion in the neck.[1,3,4] It is an artery of fair calibre and apparently may con-

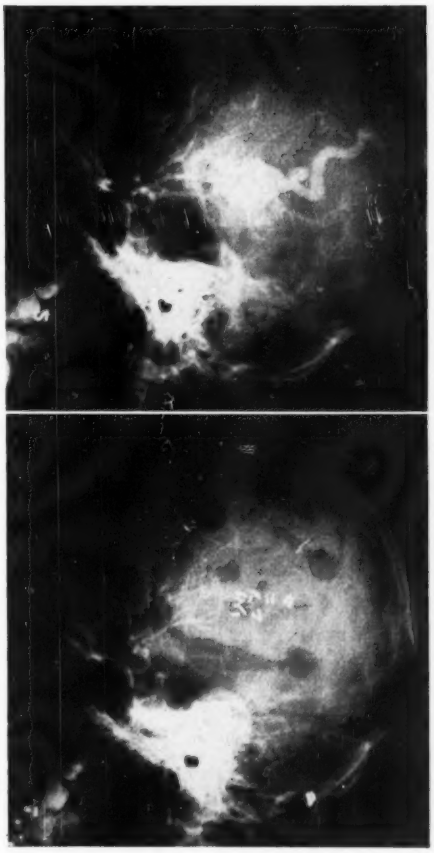

FIG. 2. (*Above*) Arteriovenous aneurysm in the parietal lobe with the appearance that all the blood from the head is draining toward the malformation. The normal vessels are very poorly demarcated as compared to those approaching and entering the aneurysm. This relative discrepancy of filling persists throughout all phases in these cases.

(*Below*) Postoperative angiogram of same case following complete excision of the aneurysm. The circulation pattern is much closer to normal.

tribute the small necessary difference between an adequate and inadequate circulation to the partially deprived hemisphere.

The arteriovenous aneurysms are less frequent and probably less lethal causes of spontaneous subarachnoid hemorrhage. They are very formidable in appearance but less dangerous surgically than the saccular aneurysms. They are best treated by complete excision. The removal of such a lesion may improve the circulation to the normal brain as it does away with the short-circuiting effect of the arteriovenous shunt (Fig. 2). The 5 arteriovenous aneurysms in this series were all treated by complete excision. There were no deaths. All patients are back at their previous occupation. The ages of this group ranged from 11 to 29 years, considerably lower than those for the saccular group, which ranged from 12 to 63.

In 22 of the 60 cases no aneurysm was visualized with bilateral carotid angiography.[2] No one of these 22 patients has had evidence of a recurrent hemorrhage to date. Two have died of other causes and there was no aneurysm found at autopsy. The vertebral system was injected in only 3 of the series. Only 5 had repeat angiograms after an interval. Some of these may have had hemorrhage on a basis other than aneurysm or in some the aneurysm may be small and thrombosed or may have been destroyed by the time angiography was performed. It is not felt that failure to visualize an aneurysm excludes such a lesion. It appears, however, that such patients are much less likely to have a fatal recurrent hemorrhage (Table 3).

The total number in this study is not sufficient to be statistically significant. In time the figures will change. However, it points out the fallacy of drawing conclusions from any number if they are grouped collectively as spontaneous subarachnoid hemorrhage. Comparisons between operative and non-operative results are not justifiable unless in addition the cases are categorized into those with proven aneurysms and those without demonstrable aneurysms.

TABLE 3

Spontaneous subarachnoid hemorrhage
(60 cases)

	No. of Cases	Age	Mortality from Recurrence (1–5 years)
No aneurysm	22	21–60	0%
Aneurysms conservatively treated	8	15–56	87%
Aneurysms surgically treated (4 operative deaths)	30	11–63	0%

SUMMARY

Sixty consecutive cases of spontaneous subarachnoid hemorrhage have been followed from 1 to 5 years after bilateral carotid visualization. In 22 cases in which no aneurysm was demonstrable there has been no evidence of

recurrent hemorrhage. The mortality in 8 cases of saccular aneurysms treated conservatively was 87 per cent from recurrent hemorrhage. In the group of 30 aneurysms treated surgically, there were 4 operative deaths. The incidence of recurrent hemorrhage in the 26 patients surviving surgery has been zero (Table 3).

It is felt that patients with spontaneous subarachnoid hemorrhage and proven aneurysm as a group are in an extremely precarious position as compared to the group without a demonstrable aneurysm. Surgery is the treatment of choice whenever possible in cases of spontaneous subarachnoid hemorrhage with a proven aneurysm. There is no lesion more benign, and yet more lethal when ruptured, than an intracranial aneurysm.

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HEMANGIOMA OF A DORSAL VERTEBRA WITH COLLAPSE AND COMPRESSION MYELOPATHY

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DURING the winter semester at the University of Berlin (1863), Virchow[12] delivered a lengthy discourse on angiomata in which brief mention was made of a patient with paraplegia who, at autopsy, was shown to have tumors composed of dilated blood channels in the center of both the 10th thoracic and 3rd lumbar vertebrae. Subsequent to this there may be found sporadic reports by pathologists concerning angiomata of osseous structures. In 1917 Hitzrot[4] recorded the first description of the roentgen appearance of hemangioma within bone. Later Perman[9] described the roentgenographic appearance of a verified vertebral angioma. Töpfer,[11] working in Schmorl's clinic, investigated at autopsy 2,154 spinal columns, disclosing a 12 per cent incidence of angiomatous masses in various areas of this structure. In a comparable study of 10,000 cases Junghanns[6] cited an incidence of 10 per cent of vertebral angiomata. These figures have recently been questioned by Lichtenstein[7] and others as being too high because some of the cases included in this study represented so-called "blood cysts." A review of the recent literature discloses reports of hundreds of cases of angiomata of the vertebrae; however, in only about 60 of these was there associated dysfunction of the spinal cord. Five of these 60 were well documented examples in which the vertebral body was partially collapsed; in the remainder the angioma was associated with compression myelopathy caused by narrowing of the vertebral canal by the enlarged pedicles and laminae. This latter phenomenon of thickened pedicles and laminae producing compression of the spinal cord in cases of vertebral angiomata has been reported by Bailey and Bucy.[1]

Only true examples of massive collapse of the vertebra with concomitant dysfunction of the spinal cord are included in the present report. The 5 cases of particular interest previously mentioned were reported by Muthmann,[8] Gold,[3] Globus and Doshay,[2] Sandahl,[10] and Holta.[5]

Muthmann's case. A 61-year-old female had progressive paraparesis of 16 years' duration. The clinical diagnosis was tuberculous spondylitis. No operative intervention was carried out because of complicating cardiopulmonary disease. Death resulted from pyelonephritis. Autopsy disclosed 50 per cent collapse of the body of the 6th thoracic vertebra with associated moderate kyphosis. Histologically there was a cavernous hemangioma within the involved vertebral body.

Gold's case. A male aged 23 had slow onset of paraplegia which was diagnosed preoperatively by roentgenograms as Kümmell's disease. At operation a hemangioma of the epidural space was found. Death followed shortly after surgical exploration. At autopsy there were found multiple lesions of the thoracic vertebrae with compression of the body of the 6th thoracic vertebra and extension of the tumor into the epidural space at this level.

Globus and Doshay's case. A 13-year-old female had a rapidly developing paraparesis of 1 year's duration which allegedly followed trauma to the back. A laminectomy was performed, following which she died. Necropsy revealed a gross collapse of the 8th thoracic vertebra which had destroyed the centrum, pedicles, and laminae, and invaded the regional epidural space.

Sandahl's case. A female, aged 17, had progressive weakness of both legs for a period of 8 years following an injury of the back. Roentgenograms disclosed compression of the body of the 12th thoracic vertebra. However, at operation an additional lesion, namely a neurinoma of the upper cauda equina, was found and removed. Although previous reviewers of vertebral angiomata have included this case, the description given by the author was not in sufficient detail to evaluate the relative significance of the two lesions. The patient survived but follow up was not reported.

Holta's case. A 66-year-old sailor had acute cervical trauma followed by gradual development of quadriparesis. Roentgenograms showed almost complete collapse of the body of the 4th cervical vertebra with a large bony fragment of the anterior inferior aspect of the body jutting anteriorly. The vertebral column was markedly angulated dorsally at the level of the lesion. Parallel striations were demonstrated radiographically within the involved vertebra and on the basis of this finding a diagnosis of angioma of the vertebra was made. After 2 weeks of cervical traction and deep roentgen therapy he died. Necropsy revealed a cavernous hemangioma of the centrum, pedicles and laminae of the 4th cervical vertebra.

Comment. In these 5 cases there was recorded a slowly developing paraparesis or tetraparesis often progressing to paralysis. It would seem there are three modes of encroachment on the vertebral canal, resulting in compression myelopathy, namely deformation of the centrum subsequent to collapse, extension of the angioma into the regional epidural space, and hypertrophy of the vertebral arch subsequent to angiomatous invasion. In some, the pathogenesis of the implication of the spinal cord appears to invoke more than one of these mechanisms. Even though the excellency of roentgen diagnosis at the present time has aided in resolving the issues in most cases, this was not always true. Many of the early reports indicated difficulty in differentiating angioma from Kümmell's disease, as well as tuberculosis.

In our clinic 4 patients with compression myelopathy secondary to vertebral angioma have been submitted to therapeutic laminectomy. No postoperative complications ensued in 3 of these. The fourth, however, had a rather rare complication, namely, collapse followed by almost complete disappearance of the centrum of the involved vertebra. The historical account of this case follows.

CASE REPORT

B.H. #134548. M.W., a 25-year-old white female, was admitted to the neurosurgical service at the Brooklyn Hospital on Oct. 5, 1948 complaining of pain in the back and weakness in both legs. She had been entirely well until March 1948 when there was pain in the left lower flank attributed to an $8\frac{1}{2}$-month pregnancy. Intermittent pain in this region persisted 7 weeks. In June 1948, after a period of remission, the pain in the left flank returned, grew more intense, and tended to radiate into the left lower abdomen. Medical advice was sought and because of the distribution of pain a pyelogram was carried out. Immediately following the pyelogram there was bilateral radicular pain arising paravertebrally and projecting into the lower anterior abdominal quadrants. Coughing, sneezing or straining aggravated the discomfort. In August there were felt for the first time numbness and tingling in both

FIG. 1. Roentgenogram on admission showing decalcification and trabeculation of the 10th thoracic vertebra.

legs and heaviness and coldness in both feet. Occasional hesitancy in starting of the urinary stream was present. Roentgenography in September was said to have disclosed no abnormalities of the vertebral column. On Oct. 4, 1948, disabling weakness of both lower extremities appeared rather precipitously, and on the following day she entered the hospital.

Examination. The head and cranial nerves were within normal limits. The upper extremities and thorax disclosed no abnormalities. The upper abdominal reflexes were equal and active, whereas the lower abdominal reflexes were absent. A Beevor's sign was present. There was marked paresis of the lower extremities. Extension of the legs, with the thighs slightly flexed, was difficult to perform. There was mild spasticity of both lower extremities, slightly more evident on the right side. The sign of Babinski was present bilaterally. Appreciation of touch and pin prick was abolished below the T11 dermatomic zone bilaterally, with minimal involvement of the sacral area. Appreciation of tuning fork vibration (c 128) was diminished at the level of the iliac crests, but absent at the knees and ankles bilaterally. Urinary retention developed shortly after entry.

Roentgenograms of the dorsal spine revealed moderate decalcification of the body of the 10th thoracic vertebra with "paralleling of the trabeculae" (Fig. 1). The lateral projection demonstrated a minimal loss of the substance of the involved centrum at its posterior inferior aspect. The intervertebral spaces were well preserved.

1st Operation. On Oct. 7, 1948, upon exposing the posterior spinous process of the 9th, 10th and 11th thoracic vertebrae, there was excessive bleeding from enlarged cortical foraminae of the lamina of the 10th vertebra. Laminectomy was attended by hemorrhage arising almost exclusively from the divided arch of the 10th thoracic vertebra. The left lamina of this vertebra was considerably thicker than that of the right side. The vertebral canal was obviously encroached upon by the enlarged pedicles of the implicated vertebra. The dural envelope was effectively released by the laminectomy. Hemostasis was secured, blood loss was replaced by transfusion and the wound was closed.

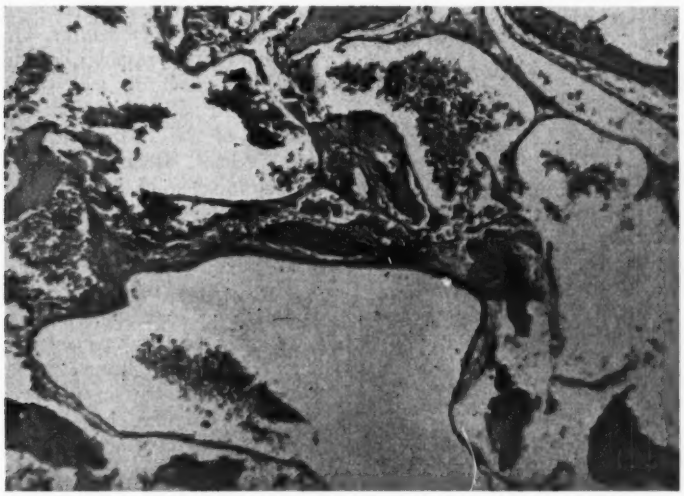

FIG. 2. Photomicrograph of the vertebral tumor, showing slender bony laminae and numerous sinusoids lined by endothelium.

Histological Report. Sections of a decalcified portion of a lamina were composed of slender bony trabeculae which were widely spaced (Fig. 2). Interspersed were sinusoids of varying size lined by endothelium and supported by scanty fibrous stroma. Diagnosis: Cavernous hemangioma of bone.

Course. Postoperatively there was gradual return of muscle power in both lower extremities. Voluntary control of the urinary bladder was reestablished on the 5th postoperative day. Ambulation occurred on the 17th day. There was no residual pain in the back or flank. She was discharged Oct. 30, 1948.

At home there was continued improvement in walking. There was occasional pain in the right thigh and the right lower abdominal quadrant. About June 1949 she noticed an increase in the "sway" of her back, and associated prominence of the lower abdomen. Without medical advice, physical manipulations of the trunk were

instituted to improve muscle tone. Shortly thereafter there was noted spasm of muscles of the back and when walking a slightly flexed posture of the trunk was assumed.

In October there was pain in the lower lumbar region with occasional radiation along both iliac crests. A week later weakness in the right leg was present followed by similar weakness in the left leg and a sensation of coldness in both lower extremities. Gradually the situation deteriorated. She was readmitted to the hospital Jan. 3, 1950.

Examination. On entry the patient walked with a halting gait and manifested weakness of both legs, more pronounced in the right lower extremity. Appreciation of position and vibration was found impaired in the right leg and foot. Touch was not felt in either lower extremity. There was diminution of appreciation of temperature below the knee on the right, and hypalgesia below the knees bilaterally. Tendon reflexes were accentuated in the left lower extremity. The sign of Babinski was present bilaterally. The healed laminectomy scar was tender. A slight protrusion of the posterior spinous process of the 12th thoracic vertebra was present. Roentgenograms of the dorsolumbar spine showed almost complete destruction of the 10th dorsal vertebra with a portion of the remaining part extruded anteriorly (Fig. 3). The lesion appeared to be limited to the 10th thoracic vertebra.

FIG. 3. Almost complete destruction of the 10th thoracic vertebra with a portion of the remaining part extruded anteriorly.

Course. She was placed on a Whitman frame with bilateral Buck's extension; however, this only served to accentuate the spinal cord dysfunction. It became evident that further surgery was necessary.

2nd Operation. On Feb. 10, 1950 the operative scar was excised. At the level of the 10th thoracic vertebra, the dura mater was encountered about 1 cm. beneath the skin. After exposing this structure, the laminectomy was increased by the removal of an arch at either end. This liberated the dural envelope. Upon incising the dura mater the full length of the wound, the spinal cord was found somewhat thinned at the level of the collapsed vertebra. The cord and its coverings were markedly displaced dorsally by an underlying dome-shaped mass which was not surgically investigated. Following hemostasis the wound was closed.

Course. Postoperatively there was immediate improvement in motor and sensory function of the lower extremities. Roentgen-ray therapy was administered. Subsequent to this a special double corset brace was provided for external stabilization. At discharge on April 4, 1950 the patient could walk with support. At this time there was impairment of appreciation of both vibration and postural movement of the lower extremities, and mild to moderate thermohypesthesia of the left leg and foot.

At home there was continued improvement in every respect. About the beginning of October 1950 she complained of the eyes feeling tired. A neurological examination disclosed bilateral moderately advanced papilledema with retinal hemorrhages. The visual fields were mildly constricted; however, visual acuity was not impaired. Small paracentral ring scotomata were demonstrated. There was no associated headache. Gradually the abnormal findings of the optic fundi receded and when examined on Feb. 26, 1951 the optic fundi were found to be normal. There was mild dysfunction of the lower extremities and rarely stress incontinence.

During the past 4 years there has been a gradual return to normal. The spinal brace has been discarded and she has been allowed to carry out all her household duties with the exception of lifting heavy objects. The most recent roentgenogram (May 27, 1954) is reproduced in Fig. 4.

FIG. 4. Roentgenogram 4 years after discharge showing recalcification of the remaining portions of the implicated vertebra and fusion of adjacent segments.

DISCUSSION

Columniation of the centrum of a vertebra is commonly interpreted by roentgenologists as hemangioma. Seldom are such lesions of clinical significance. As stated, an impressive number of vertebral hemangiomata disclosed at autopsy have been recorded and regarded as incidental findings. Although the vast majority of these lesions are innocuous, there are a few that undergo proliferative and degenerative changes. It has been proposed by Watson[13] that expansion of the tumor results from endothelial budding and subsequent canalization of the outgrowths. Cancellous bone encroached upon by these newly formed channels assumes an irregular colonnade effect or reactive trabeculae in parallelism so readily identified by roentgen examination. It has been estimated that some of the trabeculae become enlarged to as much as three times normal.

The case herein presented is rather unique because of the degree of vertebral dissolution and almost complete clinical recovery following operation. The lower dorsal region is a rather common site for the occurrence of this lesion, but it is uncommon to demonstrate anterior extrusion of a portion of a vertebral body. In light of previous experiences a complication of this character subsequent to laminectomy for compression myelopathy was not expected. The sequence of clinical events in this case serves to caution one against overestimating the strength of a vertebra involved by angioma. Fusion operations, either vertebral or rib, should be given considera-

tion. It would seem that partial collapse of a vertebral body involved by an angioma is a clear indication for a fusion operation.

The administration of roentgen therapy may help. However, it would seem unjust to recommend it preoperatively in the face of an advancing paraparesis. Certainly a judicious use of radiation for this form of lesion is indicated following laminectomy. It is to be remembered that roentgen therapy may be complicated by necrosis of normal spinal cord, especially when high dosage has been employed.

In the case herein recorded, the cause of the transitory papilledema remains obscure. Intracranial angiomatous lesions may be present although there are no other clinical features to support such an opinion.

CONCLUSIONS

Dissolution of a vertebra implicated by hemangiomatous process is a relatively rare occurrence. In the presence of clinical evidence of compression myelopathy associated with this disease, laminectomy is clearly indicated. After such an operation, roentgen studies of the involved area should be performed at intervals, particularly if there are complaints suggesting advancement of the disease within the vertebra. Furthermore, if by roentgen examination there is disclosed beginning collapse of the vertebra, a fusion operation should be performed.

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OSSIFICATION IN GLIOMAS

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CALCIFICATION of intracranial tumors has been the subject of numerous histological and roentgenological studies. Martin and Lemmen[7] studied 1577 intracranial neoplasms and found that calcification occurred microscopically in 207 cases (13.1 per cent); in 104 cases of this group calcium was visible in the roentgenogram (54.2 per cent). These authors described four different types of histological patterns of calcification, but did not mention ossification and with the exception of a case of mixed ependymoblastoma and osteoblastoma reported by Mackay[6] we were unable to find in the literature on gliomas of the central nervous system cases in which ossification had occurred. We have observed ossification in 2 cases; one was in an astrocytoma of the cerebellum of long duration (25 years); the second was in an oligodendroglioma of 9 years' duration. It is known that calcification is common in both of these types of gliomas.

Mabon *et al.*[5] reviewed 131 cases of astrocytomas of the cerebellum and found calcification radiologically in 11 cases (8.5 per cent) and microscopically in 21 (16.3 per cent). Cushing[2] reported calcification visible radiologically in only 1 of 76 cases of cerebellar astrocytomas. In neither of these papers was there mention of the presence of ossification. Earnest *et al.*[3] reviewed 200 cases of oligodendroglioma and found calcium deposits microscopically in 115 (69.7 per cent) and radiologically in 64 cases (38 per cent). There was no report of ossification.

REPORT OF CASES

Case 1. R.S., a 53-year-old white male, was admitted to the University Hospital on Jan. 3, 1955 with symptoms of severe suboccipital pain which radiated to behind the right ear, and muscle spasm in the neck which had persisted for the past 25 years. His wife stated that since their marriage years ago he had invariably complained of excruciating headaches during episodes of vomiting. Eighteen months prior to admission he had noted fatigue, increased sleepiness and blurring of vision. Six months later there developed difficulty in walking and a tendency to veer toward the left. In the last 2 months he was incapacitated.

Examination. The left pupil was larger than the right, and there was bilateral papilledema. He walked with a wide base and tended to stagger and fall backwards. There was mild decomposition of movement of both lower extremities. Deep tendon reflexes were hyperactive in both lower extremities and there was an equivocal Babinski sign bilaterally.

FIG. 1. *Case 1.* Laminagram of the skull showing a dense image of calcification in the posterior fossa.

Roentgenograms of the skull revealed an area of calcification in the midline of the occipital region, roughly ovoid in shape, that could not be seen on the lateral views. Laminagrams showed that this area was 2 cm. above the foramen magnum, immediately dorsal and superior to the fourth ventricle (Fig. 1).

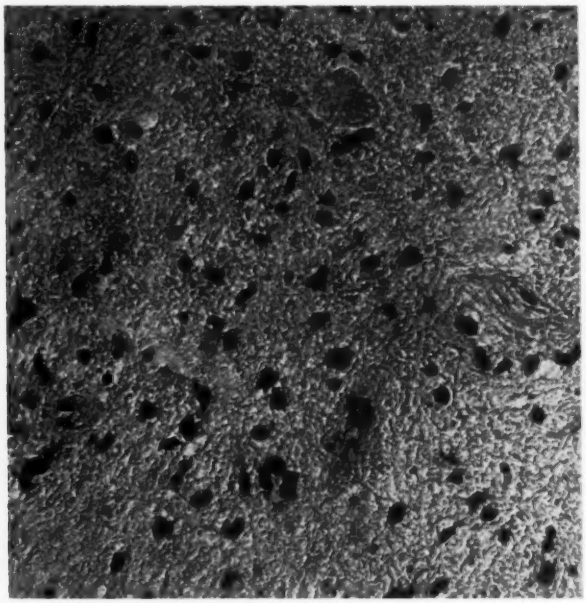

FIG. 2. *Case 1.* Photomicrograph of the cerebellar astrocytoma removed at operation.

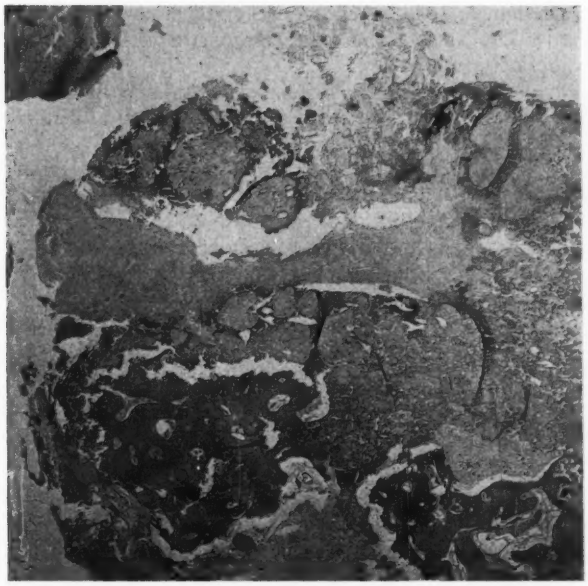

FIG. 3. *Case 1.* Photomicrograph showing calcification and ossification of the neoplasm.

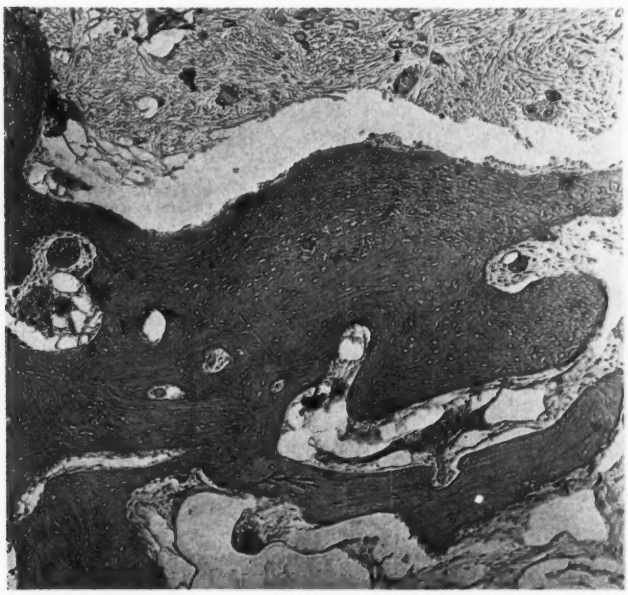

FIG. 4. *Case 1.* Higher power view of an ossified area shown in Fig. 3.

Operation. On Jan. 5, 1955, a suboccipital craniectomy and laminectomy of C1 was performed by Dr. E. A. Kahn and a large, stony-hard mass in the posterior vermis was completely removed.

Course. Convalescence was slow because of disturbance in gait and mental depression. The patient, however, was ready for discharge 1 month after operation and returned to work as an accountant 4 months later.

Histological Examination. The tumor is a fibrillary astrocytoma (Fig. 2) with hyaline degeneration and numerous calcium deposits most marked in the walls of the blood vessels. Within the tumor there is also an extensive area of calcification with irregular borders and no discernible vascular pattern. In the central portion of this calcified area there has developed well differentiated bone which contains bone-marrow spaces filled with fibrous connective tissue (Figs. 3 and 4).

Case 2. M.R., a 55-year-old white female, was admitted to the University

FIG. 5. *Case 2.* Roentgenogram of skull showing a calcified area in the frontoparietal region.

Hospital on Mar. 9, 1953. She had been well until 1946 when she had an episode of unconsciousness, generalized convulsions, incontinence of bladder and bowel, and biting of the tongue. Numerous similar attacks had occurred at irregular intervals since that time. Medication relieved the symptoms for 1 year. When x-ray examination revealed an area of intracranial calcification (Fig. 5) she was referred to the University Hospital.

Examination. An arteriogram revealed evidence of a space-occupying lesion, presumably an oligodendroglioma in the right frontoparietal region. These findings were confirmed by radio-active iodine uptake studies and an electroencephalogram.

Course. Because of the extremely slow growth of this neoplasm and the relatively minor symptomatology, surgical treatment was deferred. Phenurone was added to her anticonvulsant medication and she was discharged on Mar. 13, 1953.

Readmission, Mar. 29, 1955. She had remained relatively well until 6 weeks pre-

viously, when she became mentally confused and relatively unresponsive. At the time of admission she was semicomatose.

Examination. Blood pressure was 170/90, pulse rate 96, and respiratory rate 20. She was very obtunded, but without dysphasia; she could name objects, count numbers and add. She was fairly well oriented but extremely slow in answering questions. She could not read or write. Funduscopic examination was negative except for rather full, nonpulsating veins. There was a left supranuclear facial weakness. She had slight paresis of the left arm. There was no sensory deficit. Deep

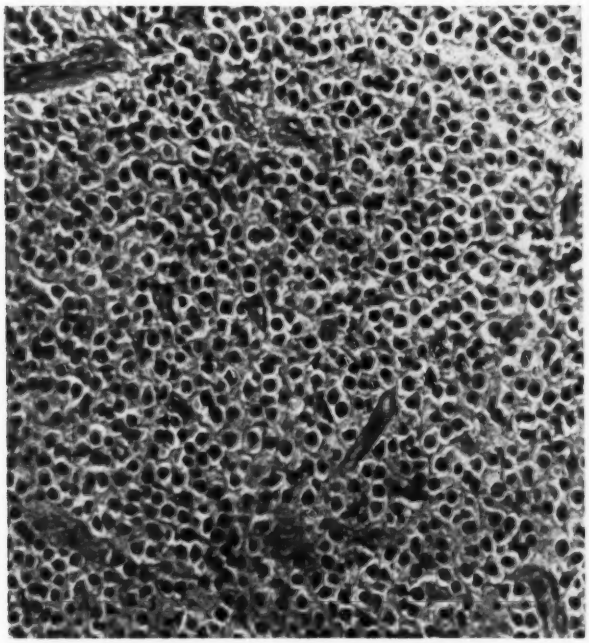

FIG. 6. *Case 2.* Photomicrograph of surgical specimen. The appearance is typical of an oligodendroglioma.

tendon reflexes were slightly hyperactive on the left with a negative Hoffmann's but equivocal Babinski sign on the left side.

Operation. On Mar. 31, 1955 a right frontal osteoplastic craniotomy was performed by Dr. E. A. Kahn and gross total removal of the neoplasm was accomplished.

Course. A few days postoperatively the mental state had returned to normal and the patient left the hospital in exceedingly good condition.

Histological Examination. The appearance of the neoplasm is characteristic of an oligodendroglioma (Fig. 6). This diagnosis is confirmed by the silver techniques. The decalcified fragments stained with hematoxylin and eosin show a deposition of calcium in concentric rings within which there is abundant membranous bone formation with typical Haversian canals and thick bony trabeculae. The bone-marrow spaces have been replaced by fibrous connective tissue (Figs. 7 and 8).

FIG. 7. *Case 2.* Photomicrograph of an area of calcification and ossification.

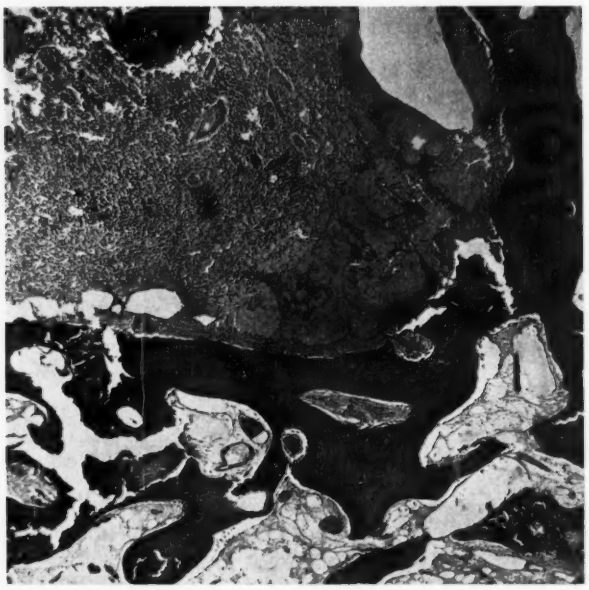

FIG. 8. *Case 2.* Detailed view of the bone formation adjacent to the neoplasm.

COMMENT

The presence of bone formation within intracranial neoplasms and particularly within gliomas arouses speculation as to the origin of the osseous tissue. It is not possible from the study of these 2 cases to determine the source of ossification within gliomas. Although we have found no discussion of the subject in the literature some possibilities should be considered as to its origin. It has been suggested in other tumors, the meningiomas for example, that bone formation occurs secondary to degeneration of tumor cells[1] or that the bone is formed by the multipotential cells that make up the tumor itself.[4] Since the tumors of the glioma group are derived from the ectoderm of the primitive neural tube, it is reasonable to assume that the latter supposition could not be applied in these cases. Although the tumors contained calcium deposits and areas of necrosis it is also difficult to accept the origin of osteoblasts from the necrotic cells themselves. Perhaps it would be more logical to assume, as did Maximow and Bloom,[8] that bone develops through a simple transformation of embryonic or adult connective tissue into a calcifiable tissue known as bone matrix (intramembranous formation). In pathological conditions, therefore, metaplastic bone formation might occur within the areas of calcified normal or necrotic tissue by direct transformation of mesenchymal cells of the blood vessels into osteoblasts, or they might arise from fibroblasts and reticular cells.

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THE SIGNIFICANCE OF TEMPORARY AND ALTERNATING PTOSIS, MIOSIS AND ANHIDROSIS*

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When the spinal cord of man is injured, a wide variety of complex patterns of activity may develop. Those such as the reflex actions of the somatic musculature,[4,9] have been the subject of many recent writings. In those patients with injury above the fourth thoracic spinal segment, hypertension and peculiar sudomotor reactions[7] result from reflex stimulation of the afferent limbs of the sympathetic system—most potently induced through the segments supplied by the sacral nerves.[8] Occasionally such reflexes may be of such a disabling nature that they require surgical elimination.[2,10] In the same general realm of interest are those sympathetic reflexes concerned with the face and eyes. A careful study has been done in regard to the ciliospinal reflex in patients with injury of the cervical spinal cord,[1] indicating that the afferent pathway is changed after injury. From all indications, the cervical sympathetic impulses operate independently of the abnormal reflex responses of the trunk and limbs.

Perhaps the most intriguing finding made in an extensive experience with patients who have had injury to the cervical spinal cord has been a condition of ptosis, miosis, and anhidrosis which has been unilateral and temporary and which could be varied by altering the position of the patient. This report deals with 6 such patients in whom explanations for the changes appear to have been found.

ILLUSTRATIVE CASES

Case 1. #159001. C.W.H., a 15-year-old boy, was injured in an automobile accident on June 16, 1951. He believes that he was able to get up to one knee, then lapsed into unconsciousness. Two days later he was moving all extremities voluntarily. Two days after this, a fracture-dislocation of C5 on C6 was manipulated, after which he was placed in traction. He did not move any extremity after this procedure. In 3 weeks he was transferred to another hospital where a laminectomy was performed.

On July 23, 1951, he was admitted to the James Whitcomb Riley Hospital with eleven decubiti, a hemoglobin of 8.5 gm. per cent, blood pressure of 100/60, and an absolute sensory and motor level at C5. He also had a urethroscrotal fistula and a severe paraphimosis.

* This paper was presented at the February 19, 1955 meeting of the Southern Neurosurgical Society held in Birmingham, Alabama.

After much supportive therapy and routine investigation, a laminectomy was carried out on Aug. 25, 1951. The spinal cord was found to be compressed laterally by bone fragments. There was a strip of yellowish tissue lying under the arachnoid for a distance of approximately 4 cm. along the right side. This was removed, apparently representing cord material that had extruded through a pial tear at the time of injury. The rent in the pia mater was found at the C6 level where a cavity was present in the cord. This tissue revealed a microscopic picture of pale-staining glial nuclei and nerve cell bodies lying in a fibrillar and loosely arranged stroma, with no evidence of inflammation. The cavity was irrigated and was seen to extend distally.

Following the operation he did very well and in the subsequent months multiple

FIG. 1. (A) Sweating test. Quinizarin powder has been sprinkled over the face. The patient has been placed on his right side for 5 minutes and then turned on his back for the photograph. The strong light has contracted both pupils. (B) One week later, the test has been repeated with the patient having been turned on his left side.

skin procedures were done for the purpose of eradicating the decubiti. After developing an automatic bladder, he was discharged in November 1951.

He was readmitted on Oct. 10, 1952, for repair of a newly formed coccygeal decubitus. At this time, his sensory level was incomplete at C6, with some scattered areas below, and he had active motor elements in the trunk. Of particular interest was the observation that when the patient was in the left lateral position, the right side of the face and neck showed obvious perspiration whereas the left side was dry. On closer examination it was also seen that the left pupil was distinctly smaller than the right, and that the left palpebral fissure was narrower than the right. When he was turned to the right lateral position, the situation was reversed. That is, the right side of the face was now dry and the left showed perspiration, the right pupil was smaller than the left and the right palpebral fissure was narrower than the left. When lying on his back, these differences were not in evidence.

Using the Quinizarin test of Guttmann[6] (Fig. 1), these differences are clearly seen. The time for development of these changes varies from patient to patient and is usually measured in minutes. It is also essential that there be a demand for sweating, although this demand need not represent a great one. Subsequently this patient has been followed further. The sign was still in evidence at the last examination.

Case 2. #126671. D.B., a 26-year-old housewife, incurred a broken neck in an automobile accident on Mar. 4, 1948. She was treated for 8 weeks with traction and then was discharged on a Stryker frame.

She was admitted to the Robert Long Hospital on Nov. 15, 1948. There was a

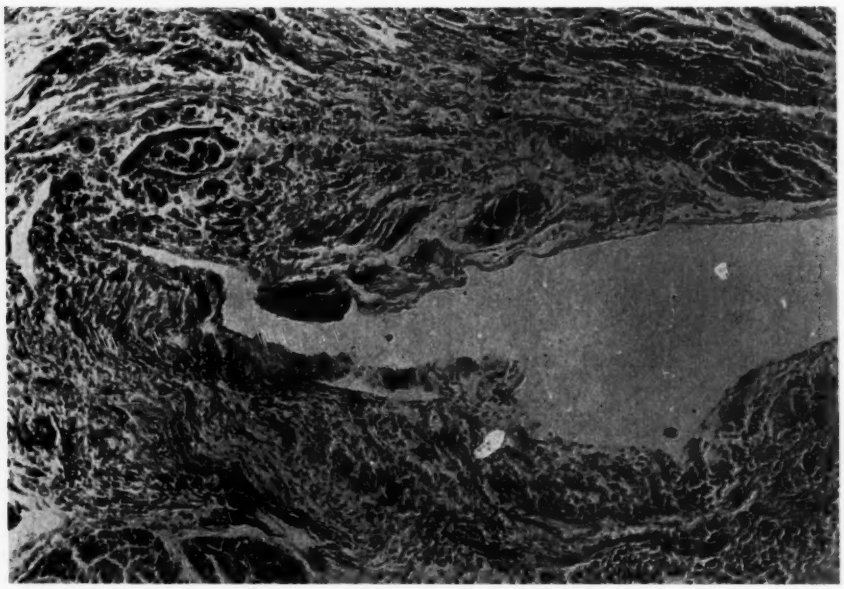

FIG. 2. Portion of wall of cystic cavity of spinal cord showing smooth cellular lining.

large sacral decubital ulcer and a complete motor and sensory loss below C5. Of particular interest was the finding that there were ptosis, anhidrosis, and miosis on the left side when she was placed in the left lateral position, no such changes when she was placed on her back, and the same changes on the right when she was placed in the right lateral position. Upon questioning, the patient stated that this condition had prevailed for several months.

Two days after admission, a myelogram was performed. It showed a large and persistent filling defect at the C4–C5 level. The findings were interpreted as a probable protrusion of intervertebral disc material.

After repairing her blood status, a laminectomy was conducted on Nov. 19, 1948, with removal of the arches of C4, C5, and C6. There were severe and dense adhesions between the spinal cord and the dura mater. These were freed. The C4–C5 disc space was protruberant and much of this material was removed extradurally.

It was noted that although the cord now pulsated, there was a darkened area at the lower end of the exposed field. This area was entered with a 25-gauge needle and about ½ cc. of viscid fluid was aspirated. The pia mater was sharply sectioned in the midline over this area and a cystic cavity was entered. The cavity was thoroughly irrigated and upon inspection, there appeared to be intact elements of white matter along the sides and bottom with the cavity apparently representing the region previously occupied by the gray matter. A small piece of Gelfoam was inserted in one end of the pial incision in an effort to keep the edges from approximating. The dura mater was closed.

She recovered from this procedure readily and in 2 weeks the sacral decubital ulcer was repaired. On numerous occasions, efforts were made to reproduce the alternating signs to no avail. She was discharged on Dec. 23, 1948.

She was followed at intervals. There was a gradual lowering of the sensory level to T1 by May 1949, with triceps function bilaterally and good radial extension. At this time it was noted that the alternating signs had reappeared. The patient despaired of life in a handicapped condition and refused to take nourishment. She died 67 days later.

At autopsy, the spinal cord was found to be lightly adherent to the dura mater. In the region of previously evacuated cyst there was a bluish discoloration, with the contour of the cord being rather constant as though it were intact. During the dissection of the cord from the canal, this zone collapsed. It could be distended by irrigation, revealing again the cystic cavity. The area collapsed upon suspension in the fixative. On microscopic examination (Fig. 2), the cord is seen to be occupied by a cavity that is lined by a layer of cells.

DISCUSSION

Four additional patients have been observed to have the alternating signs. In each of the 6 cases there were the common findings of dryness of the face, narrowing of the palpebral fissure, and constriction of the pupil on the side that was down. These were observed usually after a short period of time in the lateral position. In general, there was no detectable evidence of anything other than sympathetic balance with the patient in the supine position, although several of the patients could produce the changes by sharply turning the head and neck. When demands were made upon the body as a whole for heat regulation, the face and neck commonly were the main sources of sweating. Moreover, each of these patients had suffered similar injuries in that none of them was anatomically complete. Upon operation (in 4) it was found that the spinal cord bulged at or below the point of maximum insult. It was quite apparent that this bulging was caused by material contained within the pia mater in the central part of the spinal cord. Upon entering the bulge, a cavity containing viscid fluid was encountered. In 1 patient, the spinal cord was not bulged but the central part did contain necrotic material and there were marked adhesions between the cord and the dura mater. In Case 1 there were no dural adhesions and the cavity may not have been a closed one. The signs have been abolished in all patients after operations, returning later in 2.

Aside from the intriguing nature of the findings, a number of considera-

tions have prompted this presentation. First of these is the predictability of the lesions in each case Ordinary neurological techniques allow for accurate localization, but usually fail to give dynamic illustration of the pathological situation. These patients had in common a collection of fluid or necrotic material within the spinal cord above the cells of origin of the primary axones of the sympathetic nervous supply to the face and neck. This fluid had resulted in each instance from trauma imposed upon the previously normal spinal cord. In the one specimen that came to microscopic study, a cellular lining was seen to be present. The cystic material appears to have been residue of the degenerated gray matter of the spinal cord with possible additions through mechanisms believed to be operating in the development of other types of fluid collections. There has been intact spinal cord tissue around the cavity in each case. It is apparent from the cystic nature of the cavity that relief would necessarily be surgical in order to be permanent. But more than the simple question of abolishing a neurological curiosity would be the possible effect that evacuation of the fluid might have on the white matter in the region. Since this is the region of maximal insult, and cells in the region can be shown to be responsive, it probably follows that the less susceptible tracts could be expected to function after removal of the noxious influence. Even if the fluid under consideration were to have a specific gravity as great as that of whole blood, its total weight would still be very small. No measurements have been made of the material but its viscosity is certainly less than that of blood. This makes it all the more remarkable that actual physiological changes occur as it is shifted to bring its weight to bear on different portions of the cord. In our laboratories, we have demonstrated that minor blows to the spinal cord of animals usually result in cyst formation such as that observed here.[5] We have not yet seen a comparable group of findings in any other disorder such as suspected hematomyelia from minor injury, syringomyelia, or degenerative disorders.

In attempting to find alternate explanations of the phenomenon, one thinks of some reflex mechanisms which seem to be peculiar to the more or less isolated sympathetic system seen in many patients with injury of the cervical cord. Vascular and sudomotor effects are usually seen in these cases following manipulations of bladder, bowel, or sphincter. These responses may be of a startling nature, but they are clearly reflex and do not depend upon position. It might be that traction could be invoked to explain the findings. The principal argument against this explanation is that the majority of these patients did not have sufficient adhesions to give traction. Also, the more severely injured patients who have dense adhesions between the spinal cord and the dura mater do not show the signs. However, this explanation must be entertained in one case. To date we have not seen this phenomenon in patients with lesions that did not involve the arms, the levels having originally been from C4 to C7 by regular examination. It is probable that the lower extent of each lesion was at least at D1 since the paralysis in the arms and hands was flaccid in nature.

That the abnormal elements lying within the spinal cord are responsible

for the findings is borne out clearly by the patient in Case 2 in whom the signs disappeared after evacuation of the cyst and returned when the cyst refilled. The somatic and other sympathetic reflexes were not abolished by operation and indeed the long tracts had begun to show function, eliminating any consideration of so-called "spinal shock."

There is another point of interest that these cases bring up. Do similar fluid collections (or masses) in other parts of the nervous system exert measurable influences? It is entirely possible that masses with greater weight, even if only by infinitesimal amounts than the surrounding tissue, may produce changes only in certain positions and then only when we are prepared to detect these changes. One is instantly led to wonder about possible detection of such changes by the electroencephalogram, the pneumoencephalogram, or even plain everyday neurological examinations conducted in unusual positions.

Our original intention after seeing the first case of this phenomenon was to await an opportunity to present the findings and then defy anyone to ever find another. It was felt that here at last was an example of neurological astuteness at its zenith—except that the original observation wasn't made by us but by the patient. We intended to title the presentation "The Syndrome of Temporary and Alternating Ptosis, Miosis, and Anhidrosis" and thus come into possession of the only syndrome known which existed in only one patient and thus would never have to be learned by anyone. We hoped to attempt thereby to contribute in a small way to the discard of the many so-called convenient terms used to describe combinations of ailments, terms which are usually unpronounceable and next to impossible to remember from case to case. This would result in greater ease in teaching neurology to medical students, most of whom acquire a wholesale distaste to the discipline because of the many fancy words used. Since we have seen more cases, we are without the forceful case that we had, but we still have wholesale aversions to the use of proper names for disorders.

In patients subjected to early laminectomy and pial incision,[3] this phenomenon has not been observed, presumably because, as in the experimental material,[5] the cysts do not form once the necrotic cord tissue and blood are evacuated. In a sense, this would constitute an additional bit of evidence in favor of early operation.

SUMMARY AND CONCLUSIONS

1. Temporary and alternating ptosis, miosis, and anhidrosis occur when patients with cystic cavitation of the lower cervical spinal cord as a result of trauma are placed in the lateral positions. Upon evacuation of the cyst, the signs disappear.

2. It is postulated that the signs constitute evidence of an incomplete lesion which will benefit from surgery.

3. The question is raised as to whether it is possible to detect other lesions of the central nervous system by examination of patients in other than routine positions.

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VINYON "N" AS A DURAL SUBSTITUTE

AN EXPERIMENTAL STUDY IN THE MONKEY*

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IN THE last six decades numerous materials have been used experimentally and clinically as dural substitutes. They include metallic materials such gold,[3] platinum,[14,15] silver, aluminum, and nickel foils,[7] and tantalum and stainless steel plate or screen.[7,9,20] Biological tissues, including fascia,[13] fat,[19] periosteum,[6] amniotic, human or animal allantoic, Cargile membranes, amnioplastin,[7,8,18] gelfoam,[21] and plasma fibrin films[2,11,12] have all been used. Other substances such as rubber sheets,[1] parchment,[16] mica, cellophane,[7] olive oil, [17] polyvinyl alcohol plastic,[18] and polythene sheets,[4,5,10] have also been employed.

The chief purpose of using a dural substitute in neurosurgery is to prevent adhesion between the exposed brain and the overlying soft tissues rather than to fill the defect in the severed dura mater. It is generally believed that the meningocerebral adhesions that follow injury or surgery are responsible for the post-traumatic or postoperative convulsive disorders.

The dural substitute must possess specific qualities, such as nontoxicity, inertness in tissue, nonabsorbability, resistance to disintegration, favorable tensile strength, soft consistency, easy maneuverability for wrapping over the surface of the brain, and ease in sterilization. None of the above-mentioned materials fulfills all these requirements. This probably accounts for the long and continuous search for suitable dural substitutes in neurosurgery.

This communication concerns a new synthetic material, Vinyon "N" cloth, which has been used experimentally as a dural substitute in the monkey.

MATERIAL AND METHOD

Vinyon "N" Cloth. Vinyon "N" is a synthetic fabric woven in cloth form with a construction of 144 warps and 90 filling yarns per square inch. The woven pattern, consistency and color are those of China silk. It is nontoxic, inert, and nonabsorbable when buried in animal tissue. It has a smooth surface. Its tensile strength is similar to that of silk. Furthermore, it can readily and repeatedly be sterilized in boiling water without any appreciable damage. Autoclaving stiffens it.

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* The Vinyon "N" cloth used in this study was supplied by the Carbide and Carbon Chemicals Company, New York.

This work was partially aided by the Neurologic Research Fund of Mount Sinai Hospital, New York.

Experimental Animals and Operative Technic Used. Two series of animals were studied. The first series consisted of 6 adult monkeys including 1 *Rhesus cynomologus* and 5 *Macaca mulatta*, weighing from 2.5 to 5.8 kg.

On each of these 6 monkeys a craniotomy was performed using sterile technic. A left frontoparietal bone flap was turned down under intravenous sodium nembutal anesthesia, 18 mg. per pound of body weight. In 4 of these 6 monkeys (Animals 1, 2, 3 and 4), the cerebral cortex was left intact, while in 2 (Animals 5 and 6) a piece of the cerebral cortex in or close to the motor area was excised. In each animal, a piece of dura mater measuring approximately 1 by ½ inch was removed and replaced by a piece of Vinyon "N" cloth. The edges of the dural substitute and the dura

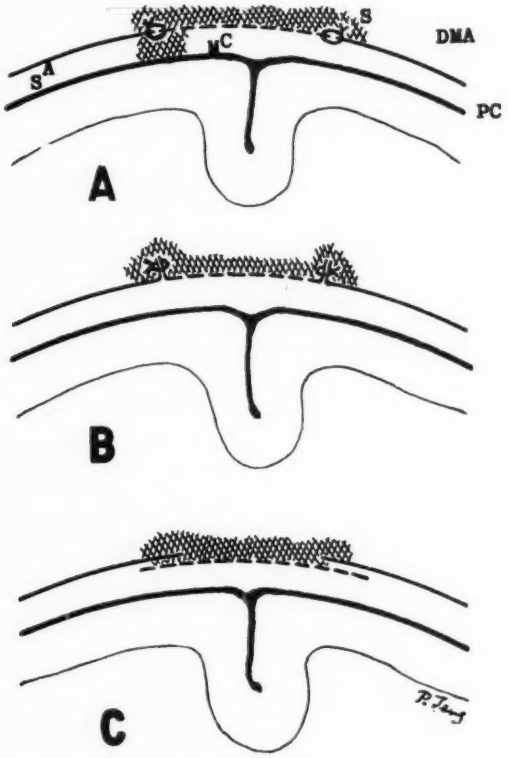

FIG. 1. Diagram illustrating the result of three different methods of anastomosis of dura mater and Vinyon "N" dural substitute. (A) Edge-to-edge (end-to-end) anastomosis with the formation of meningocerebral cicatrix along the line of approximation (crosshatch). (B) Anastomosis by everted sutures. Meningocerebral adhesions were prevented. (C) Tuck-under method shows no meningocerebral adhesions.

Broken line = Vinyon "N" dural substitute
Crosshatch = Adhesion formation
DMA = Dura mater and arachnoid
PC = Pia and cortical surface
S = Suture
SA = Subarachnoid space
MC = Meningocerebral cicatrix

mater were approximated by edge-to-edge (end-to-end) suturing with 00000 silk (Fig. 1 A) or eversion of the edges (Fig. 1 B). A second series of 5 monkeys (Animals 7, 8, 9, 10 and 11) were studied by another group of investigators for electrical stimulation of the frontal and occipital eye fields. The operative procedure employed was similar to that described above. In 3 of the 5 monkeys, the reactive area for contralateral conjugate ocular deviation was excised. In each animal a piece of Vinyon "N" dural substitute was used to cover the exposed cerebrum. The edges of the dural substitute were tucked under the edges of the dura mater with or without one through-and-through anchoring stitch on each side (Fig. 1 C). After completion of the anastomosis of the dura mater and dural substitute in each animal, the wound was closed in layers with silk sutures.

Postoperatively, each animal was observed daily until the time scheduled to be sacrificed. The animals were sacrificed after 91, 91, 380, 428, 305, 327, 21, 35, 42, 49, and 365 days, respectively.

Before each animal was sacrificed, the operative wound was re-opened under anesthesia and the situation on the outer surface of the dural substitute was first examined. Then a large dural flap which included all of the anastomosed dural substitute was reflected. The line of anastomosis, the under surface of the dural substitute, and the condition of the underlying cerebral cortex were examined. Thereafter, the animal was killed by giving it a large dose of intravenous sodium amytal. In the first series of 6 animals, the brain and its coverings as well as the dural substitute were removed en masse and fixed in 20 per cent formalin solution. After the fixation, blocks of specimen were taken from the operated area and embedded in paraffin. Sections were cut at 8μ and stained with hematoxylin and eosin, and, in some cases, with the azocarmine, phosphotungstic acid hematoxylin, Nissl, Romanes (axon) and Mahon (myelin) technics. The requirements of the physiological experiments for which the second series of animals were primarily used, precluded microscopic examination of the tissues.

OBSERVATIONS AND RESULTS

CLINICAL OBSERVATIONS

In none of these 11 monkeys have postoperative convulsions been noted. There were no abnormal neurologic findings. All animals were active and appeared normal. The wound was well healed in 10 animals and was infected in 1 (second series, Animal 9).

PATHOLOGICAL OBSERVATIONS

Animal 1 was sacrificed 91 days after the operation. The periosteum of the inner aspect of the bone flap was thickened. The dural substitute appeared grossly normal. It lay in close contact with the underlying pia and brain, so that no cerebrospinal fluid was observed. It was very easily separated from the underlying cortex, revealing only a few delicate connecting strands along the line of suture between dura mater and dural substitute. Microscopically, the periosteal and dural tissues were entirely normal except at the point of the end-to-end anastomosis. Here, fragments of suture material were noted, surrounded by a few lymphocytes and foreign body giant cells. Similar cells were present in the dural substitute, extending along its entire length. Beneath this, much more delicate connective-tissue strands of the leptomeninges were noted which revealed only a minimal proliferation of arachnoid cells. The underlying cortex appeared entirely normal.

Animal 2 was sacrificed 91 days after operation. Reflection of the dural substitute left the cortex entirely smooth. Microscopically, the dura mater at the site of the anastomosis (end-to-end) was involved in a granulomatous process like that described for Animal 1, and this process extended into the dural substitute as well. The underlying leptomeninges were thin and delicate except for two small focal areas beneath the suture line, where moderate lymphocytic infiltration and moderate fibrosis were noted. The underlying cortex appeared entirely normal, including that beneath the focal areas of leptomeningeal change.

Animal 3 was sacrificed 380 days after operation. The dural substitute was in close apposition to the cortex, but was separated from it with ease except in one small focal area along the line of end-to-end anastomosis. Here, fibrous tissues were seen extending from the epidural space and the severed edge of the dura mater through the line of suture. The adhesions were dense and firmly attached to the

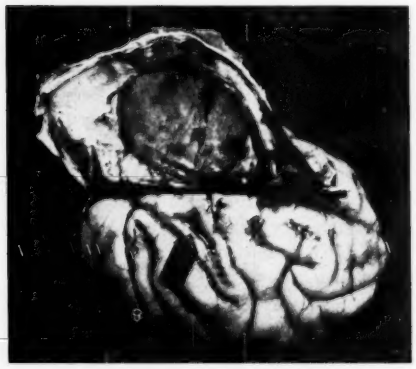

FIG. 2. *Animal 5.* Brain with reflected dural substitute shows no fibrous capsule nor meningocerebral adhesion. Arrow indicates the crator of cortical wound, surgically produced.

underlying cortex so that separation resulted in tearing. Microscopically, the point of anastomosis (end-to-end) between dura mater and dural substitute revealed a moderate granulomatous reaction like that previously described. The dural substitute contained a small number of collagen fibers, but there was no inflammatory cellular infiltration. The leptomeninges showed no essential changes. The underlying cortex was entirely normal except for the small area immediately beneath the suture line, where an adhesion to the granulomatous tissue was noted.

Animal 4 was sacrificed 428 days after operation. The dural substitute was easily separated from the underlying cortex. Microscopic sections of the anastomotic area (end-to-end) revealed a granulomatous reaction like that described above. The adjacent segments of dural substitute were similarly infiltrated and there was a dense partially hyalinized connective tissue containing a few lymphocytes and a few pigment-containing phagocytes on its inferior aspect. The leptomeninges at this site were also involved, but those beneath the dural substitute at a distance from this site revealed almost no change. The underlying cortex appeared normal.

Animal 5 was sacrificed 305 days after operation. In this case, the dural substitute was used to cover a surgical wound in the cortex. The dura mater and dural substitute were sutured after eversion of the edges, as in Fig. 1 B. The dural substitute was reflected with ease, there being no adhesions at the site of the cortical lesion, or in any other area (Fig. 2). Microscopically, a granulomatous reaction was not ob-

served on the inner aspect of the junction of dura mater and dural substitute, where this covered the brain. The external aspect was infiltrated by mononuclear and lymphocytic cells. There were no adhesions between the dural substitute and the surgical wound, which was covered by fibrotic leptomeninges showing an arachnoid cell proliferation. The wound itself showed a loss of tissue, microglial proliferation and marginal astrocytosis (Figs. 3 and 4).

Animal 6 was sacrificed 327 days after an operation identical with that used for

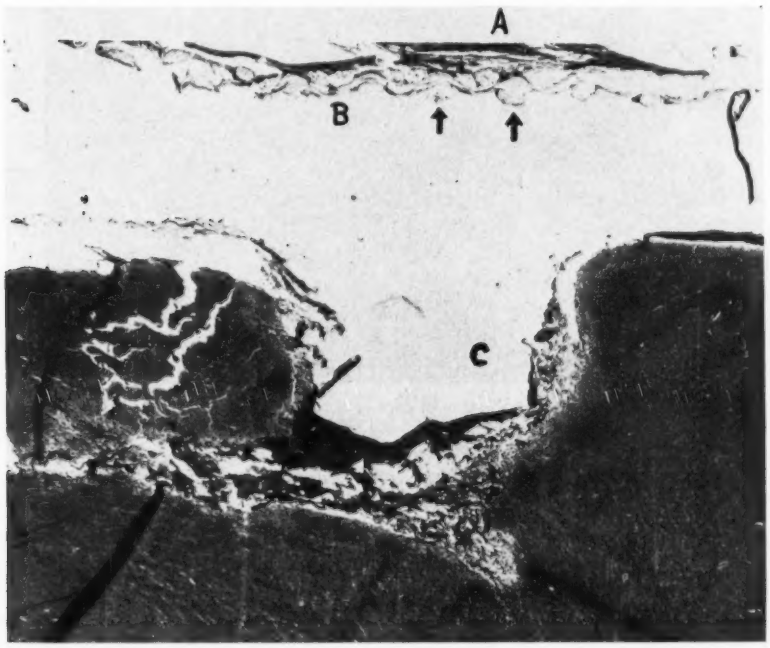

FIG. 3. *Animal 5.* The cortical wound with leptomeninges, above which is the layer of dural substitute (arrows). (A) Epidural fibrous tissue. (B) Cross-section of Vinyon "N" fibers. (C) Crator of cerebral wound. There is no meningocerebral adhesion nor fibrous capsule on the under surface of the dural substitute. Hematoxylin and eosin stain, ×26.

Animal 5. The dural substitute was easily removed, there being no adhesions at the site of the cortical lesion or in any other area. Sections through that portion of the everted anastomosis between dura mater and dural substitute in which silk sutures had been placed, revealed a small focus of granulomatous reaction like that described above. At a distance from the site of anastomosis, there were only a few collagenous fibers and no cellular reaction (Fig. 5) except at two small foci, one containing lymphocytes, the other a few foreign body giant cells. The leptomeninges were slightly thickened, but showed no inflammatory reaction except in one focal area, where a granuloma, which included giant cells, surrounded two fragments of bone wax. The cortex was entirely normal in this and adjacent areas. Sections through the brain wound, and its covering leptomeninges were inadvertently lost.

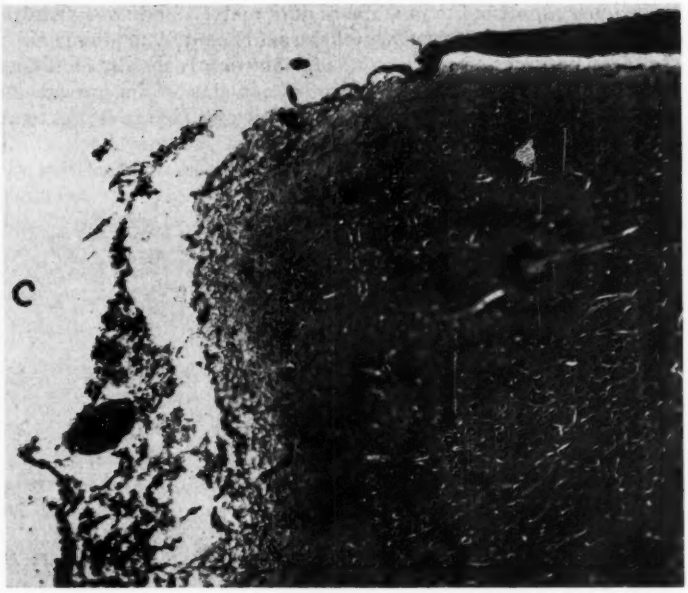

FIG. 4. *Animal 5.* Higher magnification of edge of cortical wound in Fig. 3, showing absence of meningocerebral adhesions. (C) Crator of cortical wound, surgically produced. Hematoxylin and eosin stain, ×86.5.

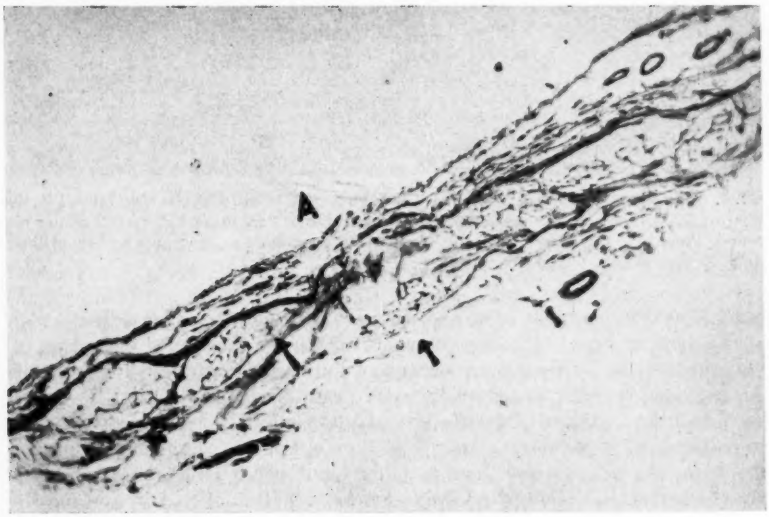

FIG. 5. *Animal 6.* Section through the dural substitute which had remained overlying a cortical lesion for 327 days shows minimal connective tissue on the under surface. (A) Epidural scars. Arrows indicate cross-section of Vinyon "N." Hematoxylin and eosin stain, ×86.5.

Observations on the second series of animals were limited to gross examination. There were no fibrous capsules about the dural substitute. There were no adhesions between the dural substitute and the cortex, including the 3 instances in which a cortical excision had been performed. In 1 animal, a wound infection with epidural abscess had developed, which extended to the outer surface of the dural substitute. When examined on the 42nd postoperative day, the inner aspect of the dural substitute was smooth, and not adherent to the underlying cortex which appeared normal.

DISCUSSION

A material to replace an excised portion of dura mater is not often required. However, it is necessary in craniocerebral lacerations, and in cases of leaking of cerebrospinal fluid through a wound connecting with the subarachnoid space. In the latter circumstance, an artificial dura mater would seal the opening and prevent leakage of the cerebrospinal fluid. In instances of penetrating craniocerebral injuries, an artificial membrane is desired primarily to reduce the incidence of meningocerebral cicatrization and subsequent post-traumatic epilepsy. It is also desired to cover the cerebral wound after the excision of a meningocerebral scar which had caused seizures.

Heavy metals induce a fibrous reaction of varying thickness and with a varying intensity of cellular reaction, when used as dural substitute. These depend on the extent and character of corrosion.[18]

Chao *et al.*[7] and Pudenz and Odom[18] have made a comparative study of various materials used as dural substitute, such as Cargile membrane, fat, aluminum, and silver foil, mica, nickel and stainless steel plate, cellophane, fascia lata, plain catgut membrane, allantoic membrane, human amnion, vinyl alcohol film, amnioplastin and tantalum foil. All were unsatisfactory. Tantalum caused the least reaction concerning the formation of fibrous tissue.

Ingraham and Bailey[11] in 1944 reported that fibrin film is easy to handle, translucent, flexible, and when used as dural substitute, was gradually replaced by a neomembrane of fibrous tissue without formation of meningocerebral adhesions. The absorption of the fibrin film was almost complete at 3 months and no film remained at 5 or 6 months after operation.

Similar results have been reported by Scheuerman and associates[21] on gelfoam film as a dural substitute. It was absorbed in 40 to 70 days after inducing the formation of a thin fibrous capsule. Nevertheless, they stated that the inner layer was continuous with the intact dura mater, making it impossible to determine the point of regeneration. There were no adhesions between the newly formed dura mater and the cortex either in the intact or in the traumatized cortex. Microscopically they found no giant cell reaction. The chief objections to these two materials are their cost, the requirement for special storage and handling, and the difficulty in sterilization.

Polythene sheet[4,5,10] has been described as the least irritating substance

among those nonabsorbable materials that have been tried as a dural substitute. Like fibrin and gelfoam film, it also caused the formation of a connective-tissue capsule, although it was thin and nonadherent to the underlying brain tissue. Its disadvantages are its nonmolding nature and its wide difference in tensile strength from the suturing material commonly used.

In our experiments with Vinyon "N" as a dural substitute, the observations were extended more than 1 year to be certain that they would reflect the ultimate degree of reaction. The evidence shows that this material placed intracranially in the monkey for a period as long as 428 days remained unabsorbed, and was comparatively inert. It did not induce the formation of a gross fibrous capsule. In 9 of the 11 animals, it completely prevented the formation of adhesions between the cortex and the overlying connective tissues even in instances in which a cortical wound had been surgically induced. In 1 (Animal 1), a few easily separable strands of tissue were noted. In the other (Animal 3) dense meningocerebral adhesions were found in one focal area along the line of end-to-end anastomosis of dura mater and dural substitute.

Microscopically, evidence of a granulomatous reaction was present about the silk sutures used to approximate the dura mater to the dural substitute, and in 2 animals this extended diffusely among the fibers of the dural substitute. In 2 other instances, fibrotic lesions were limited to that portion of the dural substitute adjacent to the anastomotic area, the distant areas being normal in 1, and showing only two small foci of inflammatory change in the other. In a fifth animal, the meshes of the dural substitute contained a few collagen fibers and inflammatory cells on its external, but not internal aspect. In the 2 animals of the first series in which a cortical wound was produced, the leptomeninges were thickened and fibrotic over the wound in 1, and most probably in the other, for which sections were not available. Other portions of the leptomeninges which lay beneath the dural substitute were entirely normal in 1, and normal except for a small focal granuloma surrounding fragments of bone wax in the other. In the 4 animals in which no parenchymal wound was created, the leptomeninges were entirely normal in 2, normal except for some granulomatous tissue beneath the anastomotic area in 1, and two small focal chronic inflammatory zones in the other. The neural parenchyma in all cases was entirely normal except for the surgical wounds produced experimentally.

These studies have shown that the method of dura mater-dural substitute anastomosis has a significant bearing on the formation of meningocerebral adhesions. Such adhesions were observed in 2 of the 4 animals in which the end-to-end anastomosis had been used. Connective tissues were seen extending from the epidural space through the suture line onto the underlying cerebral cortex in 1 of these. No such adhesions were noted in any of the other animals in which alternative methods of anastomosis had been employed. This suggests that these adhesions would have been avoided if the eversion or tuck-under technic (Fig. 1 B and C) had been substituted in

these animals. Of these two latter technics, the tuck-under method is the simpler and is the one recommended for clinical trial in using Vinyon "N" cloth as a dural substitute.

SUMMARY

1. Vinyon "N," a synthetic material in cloth form, has been used experimentally in the monkey as a dural substitute. The period of observation was 21 to 428 days.

2. This material is relatively inert and nontoxic. It is not absorbed and has a tensile strength like that of China silk. It can be sterilized by boiling in water.

3. Various methods of dura mater-dural substitute anastomosis and their relation to the formation of meningocerebral adhesions have been discussed. Meningocerebral adhesions were found along the suture line in edge-to-edge anastomosis in 2 of 4 cases in which it was used. They did not occur in any case in which suturing was done after eversion of the edges, or when the method of tucking the edge of the dural substitute under the edge of the dura mater was used.

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BEHAVIORAL AROUSAL BY STIMULATION OF THE BRAIN IN THE MONKEY*

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DURING the past five years, many data from animal experimentation have accumulated concerning the physiological mechanisms involved in sleep, arousal, and wakefulness. These observations, made on acute preparations, have indicated that appropriate stimulation of extensive but discrete areas of the central brain stem or cerebral cortex of the monkey may induce a change in the electroencephalogram identical with that observed at the onset of arousal to natural stimuli and called "arousal," "activation," or "desynchronization." Animals capable of so reacting, also exhibit movements of the eyelids and extremities suggesting that such excitation does induce a behavioral shift functionally related to that observed in arousal from normal sleep to awareness.[3,13,14]

These observations, being short-term and hence complicated by conditions of immobilization, operative trauma, artificial ventilation and other necessary concomitants of acute experimentation, require a reassessment on preparations capable of normal behavior and response. The purpose of the present experiments, therefore, was to test the effect of comparable stimulation applied through chronically implanted electrodes to monkeys examined during states of natural wakefulness and sleep. The results indicate that direct excitation of appropriate regions of the brain may indeed induce behavioral arousal of a naturally sleeping animal.

METHOD AND PROCEDURE

Experiments were made in 19 monkeys (*Macaca mulatta*) anesthetized with intravenous pentobarbital (Nembutal) and operated upon under aseptic conditions. The electrodes employed for stimulation of deep structures consisted of a pair of stainless steel wires (.005 in. in diameter and 1 mm. apart) insulated except at the tips and fastened to a thicker wire strut. They were introduced through appropriate cranial openings either stereotaxically for deep placements or under direct vision for superficial positioning. These latter placements were in the white matter immediately subjacent to the cerebral cortex.

After proper insertion, the electrodes were secured in place with liquid Kadon.

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Lead wires were brought out through a stab wound in the skin of the suboccipital region and threaded through the links of a leash for subsequent easy accessibility to a stimulator. Wounds were closed with subcuticular sutures. A detailed description of the electrode and methods of implantation for chronic stimulation has been recorded elsewhere.[7]

Eleven electrodes were implanted in deep structures and 27 in the various cortical regions described in Figs. 1 and 2. No animal had more than 4 placements and all stimulation sites were verified anatomically at the termination of the experiment.

After at least 3 days postoperatively, the animal was placed on an illuminated stage in a quiet room from which the examiner could not be seen. It was necessary to perform tests at night when the background noise was of low intensity and the animal could sleep continuously. When no reasonable doubt existed that the animal slept, as indicated by continued immobility, closed eyes and lack of reaction to noises of moderate intensity, stimulation was applied, beginning with subliminal in-

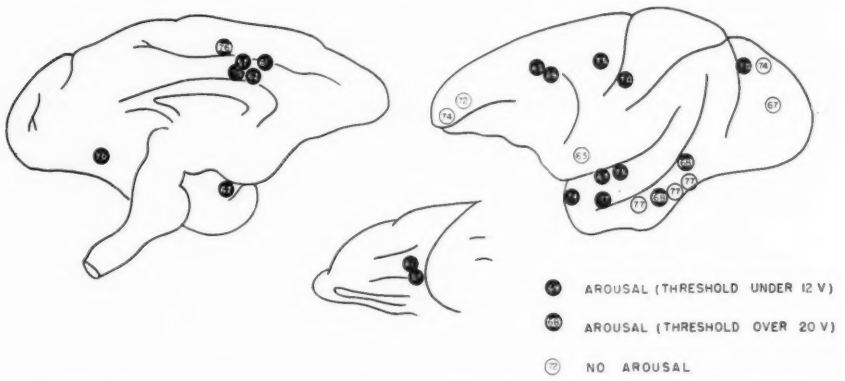

FIG. 1. "Cortical" electrode placements transferred to the right hemisphere. Black circles = arousal with intensities under 12 V. Thick open circles = arousal with intensities over 20 V. Thin open circles = no arousal. Number indicates the serial number of the animal.

tensities. The voltage was then gradually increased until the threshold for an overt response (arousal or movements) was attained or until levels of excitation that commonly induced these responses were far exceeded. Stimuli were delivered from a Grass square pulse generator (2–60 V., 1 msec., 10–300 c./sec., 1–15 sec.). Each electrode placement was tested on from 5 to 15 different nights for a period of up to 4 months after implantation. It was possible, therefore, to record typical responses photographically. Similar excitation was applied to the animal when it was awake.

In order to assess, simultaneously, the EEG and behavioral changes associated with arousal, observations were made upon 2 animals (#76, #77) with electrodes placed in cortical and subcortical loci. Sites selected were the reticular formation, the superior temporal gyrus and the cingulate gyrus. All electrode placements in these experiments were paired so that the area of excitation could be monitored electroencephalographically for afterdischarge. Additional EEG recordings were made in these animals from points in the brain stem and cerebral cortex distant from the stimulated site. Needle contacts were inserted into the skull to record activity on both sides of the cranial vault.

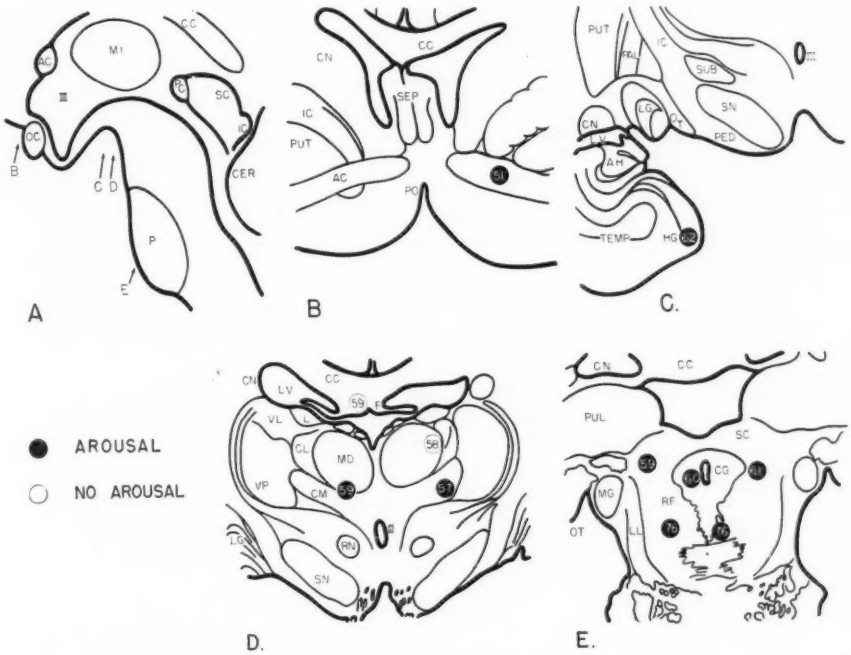

FIG. 2. (A) Midsagittal section of brain stem showing levels of frontal sections (arrows). (B, C, D, E) Frontal sections of brain with deep electrode placements. Black circles = arousal. Open circles = no arousal. Number indicates serial number of experiment.

AC = anterior commissure
AH = Ammon's horn
CC = corpus callosum
CER = cerebellum
CG = central gray
CL = centralis lateralis nucleus of thalamus
CM = centre median nucleus of thalamus
CN = caudate nucleus
F = fornix
HG = hippocampal gyrus
IC = internal capsule
L = lateralis nucleus of thalamus
LG = lateral geniculate nucleus
LL = lateral lemniscus
LV = lateral ventricle
MD = medialis dorsalis nucleus of thalamus
MG = medial geniculate nucleus
MI = massa intermedia
OC = optic chiasma
OT = optic tract
P = pons
PAL = globus pallidus
PED = cerebral peduncle
PC = posterior commissure
PO = preoptic region
PUL = pulvinar
PUT = putamen
RF = reticular formation
RN = red nucleus
SC = superior colliculus
SEP = septum
SN = substantia nigra
SUB = subthalamus
TEMP = temporal lobe
VL = ventralis lateralis nucleus of thalamus
VP = ventralis posterior nucleus of thalamus
III = third ventricle

RESULTS

Stimulation of the Sleeping Animal. Excitation of appropriate areas of both the cerebral cortex and brain stem resulted in a response in which the animal opened its eyes, shifted the position of its head, limbs and body and

FIG. 3. Behavioral effects of stimulation of central nervous system on a sleeping monkey. The excitation of certain areas (reticular formation, temporal pole) may induce arousal with reasonably low intensities (2–8 V.) whereas that of others (e.g., frontal pole) does not, even with high voltages (45 V.). Each vertical column shows pictures of a single stimulation. Top frame, pre-stimulation control of sleeping animal (PRE.-ST.); 2 central frames, during the stimulation (ST.) indicated by lighted rubies on back of cage; lower frame, following stimulation (POST.-ST.).

FRONT. P. = frontal pole. RF = reticular formation (mesencephalic gray). TEMP. P. = temporal pole. V. = volts.

reacted to auditory and visual stimuli as though alert to and adequately oriented in its environment (Fig. 3). This "arousal" usually resulted immediately upon application of the stimulus and, if it was to occur, did so within a few seconds. While minor variations in the appearance of the induced response occurred on different occasions, the general nature of the reaction was strikingly similar for all active loci regardless of the animal tested or the site stimulated. When a slightly higher voltage was employed, the animal would awaken and look about searchingly in what seemed a puzzled manner. With some electrode placements, further increases in intensity of the stimulus induced a very abrupt "arousal" together with "crouching" or "flight" reactions that will be described later.

These results could be obtained consistently by excitation of the mesen-

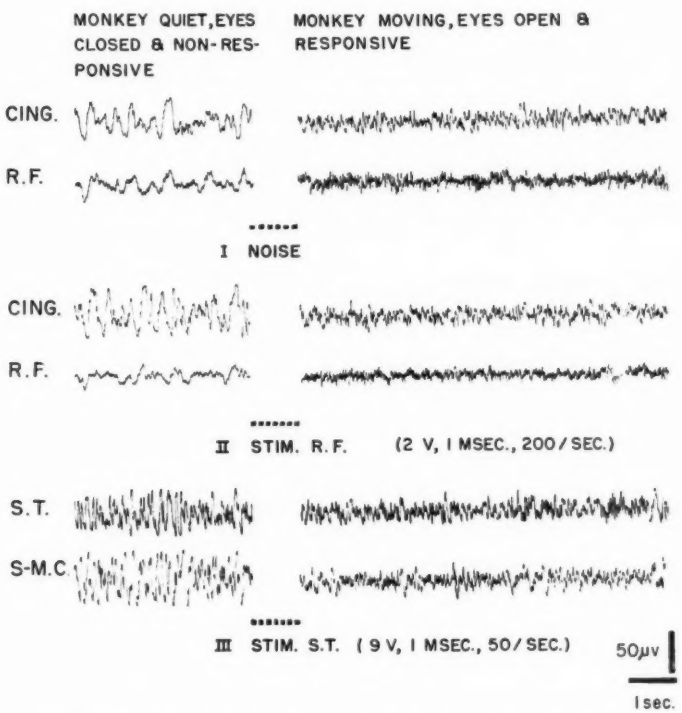

FIG. 4. Concomitant effects of arousing stimuli on behavior of animal and on electrical activity of its brain: I, noise; II, electrical stimulation of reticular formation; III, electrical stimulation of superior temporal gyrus. During the high voltage and slow activity (tracings on the left), the animal appeared to be asleep. After the arousing stimuli had been applied, the animal exhibited a low voltage and fast activity of cortex and of brain stem (tracings on the right) and was awake. The tracings from R.F. and from S.T. were recorded in the immediate vicinity of the stimulating electrodes.

CING. = cingulate gyrus. R.F. = reticular formation (mesencephalic central gray). S-M. C. = sensorimotor cortex (contralateral to stimulation region). S.T. = superior temporal gyrus.

cephalic central gray substance and reticular formation, centre median of the thalamus, hippocampal formation, anterior commissure, orbital surface, cingulate and first temporal gyri, temporal pole, para-occipital area, and basal surface of temporal lobe (Figs. 1 and 2).

Excitation of the frontal oculomotor field also induced arousal which was generally preceded by adversive displacements of head and body. With intermittent short bursts of stimulation (13 V., 1 msec., 50 c./sec.) it was possible to induce arousal similar to that initiated by excitation of other active regions, without adversion or other motor manifestations which in themselves might have initiated wakefulness. Motor concomitants of arousal in the form of Jacksonian seizures resulted from excitation of the motor cortex (or, with higher voltages, the primary somesthetic region) with stimuli of 5 to 12 V., 1 msec., 50 c./sec. Here, however, it was impossible to resolve the two events (arousal and movements) by modification of the stimulus parameters.

As may be seen in Fig. 4, behavioral arousal induced by noise or by excitation of effective cortical or subcortical loci was accompanied, consistently, by a generalized shift of the EEG from high voltage, slow activity to a low voltage and fast pattern. Intensities that were effective in this respect did not induce any abnormal activity (e.g. afterdischarge) in the vicinity of the stimulating electrodes.

Though it was possible only in a general way to assess the depth of sleep of the animal at the time of stimulation, it was apparent that drowsing animals could be aroused more promptly and with lower voltages than could those monkeys that were deeply asleep. Even under such circumstances, however, stimulation of effective loci always resulted in the awakening of the animal and, on different nights, the same preparation responded to very similar threshold intensities.

No arousal or other visible effects resulted from stimulation as high as

TABLE 1

Summary of effects obtained by stimulating the brain stem

Area	Nos. of Animals	Response	
		Asleep	Awake
Reticular formation	59, 60, 61, 75, 76	Aroused 2–5 V.	Alerted 2 V. Cowed, flight reaction 3+ V.
Centre median nucleus of thalamus	57, 59	Aroused 3–4 V.	Alerted 3 V. Cowed, flight reaction 5+ V.
Anterior commissure	57	Aroused 9 V.	Adversive seizure 10 V.
Fornix	59	Not aroused 15 V.	Nothing with 15 V.
Medialis dorsalis nucleus of thalamus	58	Not aroused 10 V.	Not tested

30 V. in the deep structures and as high as 60 V. in the cortical areas marked as open circles in Figs. 1 and 2. The lower premotor zone was considered negative because no arousal ever resulted from stimulations that consistently induced Jacksonian seizures of the face (8 V., 1 msec., 50 c./sec.).

Threshold values for voltages inducing arousal are listed in Tables 1 and 2. In general, lower values were required to initiate this effect from mesen-

TABLE 2

Summary of effects obtained by stimulating the cortex

Area	Nos. of Animals	Response	
		Asleep	Awake
Orbital	66, 67	Both aroused 6–7 V.	Alerted to contralateral side 7 V.
Frontal pole	72, 74	Adversive seizure with no arousal 40–45 V.	Adversive seizure 25 V. (72); nothing 50 V. (74)
Gyrus cinguli	76, 57, 61, 62	Aroused 6–10 V.	Alerted 7–9 V.
Area 8	63, 69	Adversive movements 12–13 V. with or without arousal; aroused without adversive movements only on intermittent stimulation 13 V.	Adversive movements 10–13 V.
Premotor area (low)	63	Masticatory movements, Jacksonian seizure 16 V.; not aroused	Masticatory movements, Jacksonian seizure and torsion 15 V.
Motor area (arm)	73	Jacksonian seizure and aroused 8 V.	Clonic jerks 5 V.
Sensory area (arm)	70	Jacksonian seizure and aroused 8 V.	Paresthesia (?) hand and face 7 V.; Jacksonian seizure 12 V.
Para-occipital area	70	Aroused 6 V.	Alerted to contralateral side 6 V.; cowed 8 V.
Occipital lobe	67, 74	Not aroused 40 V.	Nothing (67) or searches floor on contralateral side and rubs eyes 10 V. (74).
Basal surface occipital lobe	70	Aroused 6 V.	Not tested
Temporal tip	74	Aroused 9 V.	Alerted to contralateral side (sometimes homolateral) 14 V.
1st temporal gyrus	69, 73, 77	Aroused 12 V.	Alerted to contralateral side 12–15 V.
2nd temporal gyrus	68, 77	Aroused 20–22 V.; not aroused	Alerted to right 24 V.; nothing
Gyrus hippocampus	62, 63	Aroused 5 V. (63 not tested)	Alerted 12 V.; clonic movements of hand 13 V. (62 not tested).

cephalic and diencephalic placements. Most cortical areas exhibited thresholds of from 5–12 V.; the lowest values were encountered in the hippocampal formation, the cingulate gyrus and the orbital surface, while somewhat higher intensities had to be applied to the posterior parietal and first temporal placements. In one instance, excitation of the second temporal gyrus occasionally aroused the animal when values of 20 V. had been exceeded. The results obtained from this area, however, were mainly negative.

A systematic study of the effectiveness of different frequencies of stimulation was difficult, as animals, once aroused, might stay awake for prolonged periods. Occasionally, a preparation slept soundly and regularly and, in these instances, tests showed the effective frequencies to be 50–300 c./sec. in the brain stem and 20–300 on the cortex; the most effective frequencies (as judged by lower threshold voltages) ranged between 100–300 and 50–200 c./sec. respectively.

Stimulation of the Waking Animal. The effects obtained in waking animals are summarized in Tables 1 and 2. During wakefulness, excitation of areas that aroused the preparation from sleep commonly caused the animal to stop whatever spontaneous activity was in progress, to raise its head, retract its ears, and become alert as if in preparation to react promptly to some unknown threat (Fig. 5). Searching movements of the head and the eyes were performed in an inquisitive manner. These movements were directed to both sides when induced from the brain stem, but were directed more commonly to the contralateral side upon cortical stimulation. During this period, the animal still responded appropriately to other stimuli and appeared to retain adequate awareness of and reactivity to its environment. This "alerting" reaction did not occur if the animal was already behaving excitedly in response to the proximity of something or somebody that it seemed to consider alarming. Contrastingly, the reaction became most obvious when the animal was quiet. This response was reproducible in individual cases at very consistent threshold voltages that generally were equal to or slightly higher than those that induced arousal from sleep.

While the response to liminal stimulation suggested "alerting," that to higher voltages assumed additional features simulating an emotional response such as fear (Fig. 5). This behavior was particularly clear and consistent when excitation was applied to the mesencephalic central gray or reticular formation or to the centre median. With intensities of stimulation higher than those yielding "alerting," the animal would cringe as if in preparation for defense or flight ("cowering"). Still greater intensities of stimulation would cause the animal to race agitatedly about the stage in what appeared to be an effort to escape an unknown but impelling danger ("flight").

In addition to these common behavioral responses of "alerting," "crouching," and "flight," elicited from diverse cortical and subcortical regions, certain reactions peculiar to the excitation of individual areas occurred. On stimulation of the hand region of the somesthetic cortex (7 V., 1 msec., 50 c./sec.), the animal would look at the contralateral extremity and rub or

FIG. 5. Behavioral effects of stimulating the mesencephalic central gray on an awake monkey. By increasing the intensity, the alerting (3 V.), cowering (4 V.) or flight (6 V.) reactions may be obtained. Each vertical column shows pictures of a single stimulation. Top frame, pre-stimulation control (PRE.-ST.); 2 central frames during the stimulation (ST.) indicated by lighted rubies on back of cage; lower frame, following stimulation (POST.-ST.).

massage the fingers with the unaffected upper extremity; an increase in voltage to 12 V. resulted in a Jacksonian seizure of the contralateral hand. Similar motor seizures would occur upon exciting the motor cortex (5 V., 1 msec., 50 c./sec.). On excitation of the external surface of the left occipital region (10 V., 1 msec., 50 c./sec.), the monkey would blink repeatedly looking downwards and to the contralateral side, rub its eyes or pass its hand over the floor in a searching manner. Stimulation of the frontal oculomotor

fields in the waking animal elicited adversive movements similar to those described in the sleeping animal.

DISCUSSION

Direct evidence concerning neural mechanisms involved in the initiation of wakefulness has been derived, so far, from acute experiments performed on curarized, anesthetized or *encéphale isolé* preparations.[3,13,14] Two main criteria of arousal have been utilized: on the one hand, the transformation of the EEG tracing from the high voltage, slow record of sleep to the low voltage, fast activity characteristic of wakefulness; on the other, in *encéphale isolé* or lightly curarized preparations, the initiation of movements of eyelids, body or extremities. While deductions based upon these observations may strongly suggest that stimulation had induced true arousal, it was felt that any conclusions would have added significance if founded upon behavioral responses of chronically prepared and normally sleeping animals. The complicating factors of recent operative trauma, immobilization, administration of anesthetic drugs or curare and of exposure of the brain would thus be eliminated. Moreover, observations on such unencumbered preparations would make possible evaluation of any motor or even sensory concomitants which in themselves might induce arousal.

The results of the experiments reported here confirm earlier studies in which EEG "desynchronization" and rudimentary behavioral responses were used as criteria of arousal in acute preparations. Behavioral arousal could indeed be induced by suitable stimulation of discrete loci within the brain stem or on the cerebral cortex. Areas from which positive responses (arousal) were obtained were identical in both studies. Moreover, in both groups of experiments effective threshold voltages were comparable and optimal frequencies the same. When behavioral and EEG changes were observed simultaneously in chronically prepared sleeping animals, awakening and EEG "desynchronization" occurred simultaneously upon the application of a natural (noise) or artificial (electrical excitation of an active locus) arousing stimulus.

Previous observations in acute experiments have indicated that a stereotyped arousal response could be induced by excitation of sensory systems and of discrete but widely separated cortical loci. It was also proposed that this response was mediated by convergence of influences upon a diffusely projecting system of neurones in the central cephalic brain stem.[3,6,13,14] Supporting this proposal were the facts that excitation of the reticular activating system provoked EEG arousal and that its destruction resulted in a preparation that displayed irreversible EEG synchrony and persistent coma. The observations reported here strengthen these concepts by adding positive behavioral evidence to deductions based so far on acute stimulation or chronic lesion studies.

The appearance of the arousal response in these normally sleeping animals was identical whether the liminal stimulus which initiated it was applied to peripheral end organs, to the brain stem or to the cortex. Jack-

sonian movements occurred only when the motor cortex or the frontal oculomotor fields were excited and, in the latter case, were easily resolved from the arousal response by modification of the stimulus. No animal, upon being awakened by current applied to any active area, appeared to have experienced a painful or startling sensation. With a single exception (sensorimotor cortex), therefore, arousal to threshold stimulation of active loci was not associated with manifestations that could lead to the interpretation that arousal was secondary to a primarily induced motor or sensory phenomenon.

In the waking state, stimulation of active zones with current intensities equal to the threshold for arousing the sleeping preparation, commonly induced no change in the behavior of the monkey. The application of slightly supraliminal stimuli to these regions, however, resulted in a performance suggesting that the animal had become "alerted," as if surprised by a natural stimulus. This "alerting" reaction, undoubtedly related to arousal, resembles closely that described by Gastaut, *et al.*,[8] MacLean and Delgado[12] and Kaada, *et al.*[10,11] after stimulation of rhinencephalic areas. Stronger stimulation of active zones evoked behavioral patterns comparable to those naturally associated with states of fear and agitation; when extreme, the reaction clearly typified the frantic efforts of monkeys to escape a situational challenge. From what is already known of the physiology of these areas, it is evident that the same regions of the brain that cooperate in the initiation and maintenance of alert wakefulness, may be involved in initiating visceral[15] and somatic responses that are known to appear normally under menacing, startling or stressful situations.

In addition to data pertinent to arousal, the results obtained from stimulation of the primary cortical areas for vision and somesthesia may have some bearing upon general problems of sensation and perception. During wakefulness, excitation (10 V.) of the visual area evoked behavior that suggested that the animal had experienced visual sensations. Yet comparable or far more intense stimuli (60 V.) applied to the same cortical locus of the monkey during its natural sleep evoked no response whatsoever. Similarly, a stimulus of low intensity (7 V.) applied to the somatic sensory area of a wakeful animal initiated activity suggesting that paresthesias were perceived in the contralateral hand. When applied during sleep, however, much more intense stimulation (12 V.) was required to awaken the animal and such arousal always was associated with Jacksonian movements. In order for a stimulus to be perceived, therefore, it would seem that the excitation present in primary systems must either find or initiate a condition of alert responsiveness in the animal. The disparity between thresholds for induction of arousal and of apparent perceptive experience by stimulation of the primary sensory areas tested would support the thesis that these two phenomena, intimately related functionally, are mediated by different mechanisms. Information exists that indicates that the alert or receptive state is induced or mediated by extralemniscal systems.[1,2,4,5,13]

It is evident that neural processes underlying wakefulness, "conscious-

ness," and sleep are of the utmost importance in an understanding of human behavior. While such phenomena can be explored directly in the experimental laboratory, it is difficult to know the extent to which data so derived will support clinical interpretation. Extrapolation must be particularly cautious when made on the basis of acute observations on animals as reliance can be placed only partially on gross anatomical approximations of the subhuman primate and human nervous systems. There are general behavioral similarities in animal and man, however, such as those displayed in sleeping, arousing, attending to stimuli, etc. and it is possible that the long-term experimental observations on monkeys reported here strengthen concepts that suggest that comparable mechanisms are involved in humans.

Additional information exists which supports the applicability of laboratory experimentation to a study of function and disorder of the nervous system in man. Recently, Hassler[9] demonstrated arousal of a lightly anesthetized sleeping girl by a stimulus delivered directly to the central cephalic brain stem, a region identical with that from which a similar response was induced in monkeys. Moreover, the pathological counterpart of wakefulness, coma, has been shown to result from destruction of this same brain stem area in man and animal. Such parallels in response of the two species to comparable test conditions serve to indicate that the experimental laboratory has much to offer the clinic in studies of the nervous system.

SUMMARY

In monkeys with chronically implanted electrodes, cortical or subcortical regions of the brain have been stimulated during spontaneous sleep or wakefulness.

Behavioral arousal from natural sleep can be induced by excitation of regions of the central cephalic brain stem and cerebral cortex which, in earlier acute experiments, proved to be effective in inducing "desynchronization" of the electroencephalogram.

In the waking monkey, liminal stimulation of the same regions causes "alerting" reactions and more intense excitation induced "cowering" or "flight" responses. Observations pertinent to the problem of perception and its apparent reliance upon the alert wakeful state were made.

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REPORT ON 280 CASES OF VERIFIED PARASAGITTAL MENINGIOMA

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DURING the last three decades, from 1924 through December 31, 1954, 280 cases of verified parasagittal meningioma have been observed at the Serafimerlasarettet in Stockholm.

A first report on such tumors was given in a monograph in 1934,[3] and the cases up to 1946 were summarized in a second pertinent publication in 1947.[4] Since then, 75 new cases have been observed, and it seems appropriate at this time to report on the entire series of the past 30 years.

General Statistics of Intracranial Tumors. The total number of verified

TABLE 1

Verified brain tumors up to December 31, 1954

Diagnosis	No. of Cases	Per Cent
Gliomas	2437	46.5%
Meningiomas	1004	19.2%
Neurinomas	420	8.0%
Adenomas	445	8.5%
Angiomas	129	2.4%
Arteriovenous aneurysms	161	3.1%
Arterial aneurysms	204	3.9%
Craniopharyngiomas	92	1.7%
Cholesteatomas	37	0.7%
Teratomas, Chordomas	17	0.3%
Papillomas	17	0.3%
Granulomas	55	1.0%
Metastatic tumors	179	3.4%
Miscellaneous	53	1.0%
	5250	100.0%

brain tumors observed in this department up to December 31, 1954, is 5250. Of this total number, 1004 were meningiomas. The classification and percentage of all tumors is given in Table 1.

Localization of Parasagittal Meningiomas. Table 2 shows the localization of parasagittal meningiomas. In subdividing this meningioma group the principles discussed in the monograph of 1934[3] have been applied.

Again the large number of meningiomas of the falx is of interest, the reason probably being the same as discussed previously. Reports from other clinics also indicate that falx meningiomas are much more frequent than

TABLE 2

Localization of parasagittal meningiomas

Location	No. of Cases
Sinus longitudinalis superior	
Anterior third	57
Middle third	111
Posterior third	31
Falx	81
	280

was assumed by Cushing.[1] Recently, Petit-Dutaillis and Pertuiset[5] found 16 meningiomas of the falx among 74 meningiomas of the cranial vault.

Age and Sex Distribution. The distribution of parasagittal meningiomas in relation to the age of the patient on admission is shown in Fig. 1. The peak is between 45 and 55 years. The sex distribution was evenly 50 per cent male, 50 per cent female.

Symptomatology. A description of the signs and symptoms of this tumor group appears unnecessary. We refer the reader to other publications in which this subject is extensively discussed.[1,3] Of greatest importance for the diagnosis are the roentgenological findings and the length of history, which are presented below in more detail.

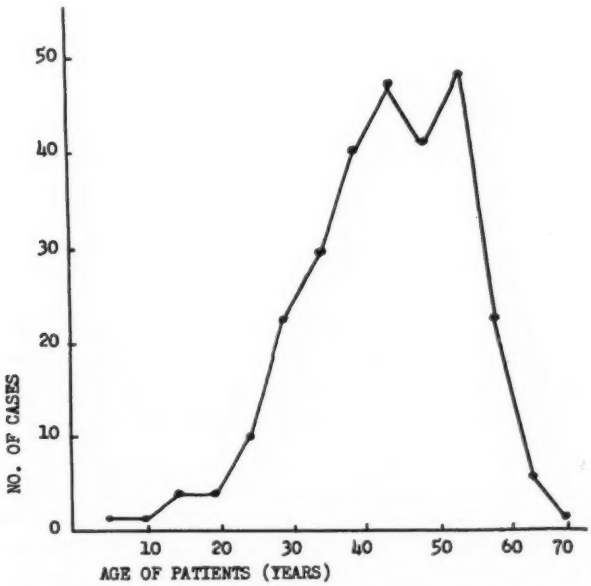

FIG. 1. Age distribution in 280 cases of parasagittal meningiomas.

Roentgenological Findings. Specific changes considered characteristic of meningiomas were present in 71 cases, or 25.4 per cent. Lindgren[2] mentioned the occurrence of bony changes in 25 per cent. The most common changes, according to Lindgren, are enlargement of the canaliculi of Havers, regeneration and sclerosis of bone, and periosteal bone regeneration with formation of spicula. Calcifications are infrequent. Changes in the vascular channels and in the size of the foramen spinosum are often present but difficult to evaluate. In 45 to 50 per cent of Lindgren's material (which is mostly derived from patients seen or operated upon in our department) abnormal vascularization, enlarged arterial or venous sulci (without localized bony changes), signs of increased intracranial pressure or displacement of the pineal body were noted. Meningiomas of the falx often produced pressure signs in the region of the sella; these were present in 6 of 10 cases in Lindgren's series. Among 19 cases of meningiomas of the falx observed here since 1947, 8 showed x-ray evidence of increased intracranial pressure, and 2 showed pineal displacement. Calcification was present in only 1 of these 19 cases.

Length of History. This information is, next to the specific roentgenological changes, of eminent help in establishing the proper diagnosis. In 172 cases the duration of history was 1 year or more and in 100 it was over 2 years. In 5 of the 75 cases observed from 1947 through 1954 the history was of 10 or more years' duration. On the other hand, a relatively short history of less than 6 months was obtained in 56 cases, and of less than 3 months in 17 (curiously enough 4 of these, almost 25 per cent, were seen in 1954 alone!). We are presenting here 2 examples of an unusually short history.

Case 76/54. A 43-year-old female had always been in good health until she suffered a skull trauma on Jan. 10, 1954, involving the right parietal region. She was dazed for a few minutes and complained of severe headache. There were no abnormal neurological findings. Forty-eight hours later there was elevation of temperature, and she had pain and paresthesias in the left arm, progressing into complete left-sided hemiplegia, with left facial paralysis and left-sided anesthesia. Vomiting occurred. She was admitted to the hospital 5 days after the accident. Encephalogram and arteriograms revealed a right-sided parasagittal tumor, and patient was referred to the Serafimerlasarettet. At operation on Jan. 29, 1954, a parasagittal meningioma of 52 grams was removed from the right motor area.

Case 549/54. A 46-year-old man, formerly of good health, noted occasional feelings of pressure in the head and some headache during the last few months. In May, 1954, he suffered two episodes of loss of consciousness. In June, 1954 angiogram and encephalogram demonstrated a right frontoparietal tumor. At operation a right parasagittal meningioma the size of a tangerine was found in the middle third of the sinus.

Contrast Roentgenography. With exception of 13 early cases in the period of 1924 through 1930, contrast roentgenography has been used in all cases, even those in which the diagnosis appeared to be unquestionable. The main reason for this practice is to determine as accurately as possible the size and extent of the tumor in order to make the bone flap of appropriate size.

Encephalograms were performed in 118 cases, and *ventriculograms* in 132 cases, including those cases in which encephalography gave insufficient filling. As a general rule encephalograms instead of ventriculograms were carried out whenever no signs of increased intracranial pressure were present. *Arteriograms* were used in 90 cases. First used in a case of parasagittal meningioma in 1932, this method has been applied more and more frequently ever since. Table 3 demonstrates the contrast studies used in our cases of parasagittal meningiomas during various periods.

The increase in the number of arteriograms since 1932 is sufficient proof for the usefulness of this particular method. In fact, the use of angiograms in recent years has been so constant that among the 53 patients operated upon since 1949 there were only 3 who did not undergo preoperative angiography. In a great number of cases, angiography was combined with encephalography. In contrast, ventriculography has become considerably less frequent in the last decade. A detailed discussion of the factors determining this change in diagnostic methods and of the advantages of the various contrast methods is beyond the scope of this publication. Angiography frequently makes a positive diagnosis of meningioma possible. It also gives valuable information concerning the vascularity of the tumor. Injection of the sagittal sinus might give information concerning the patency of this structure but so far we have not thought it worth while using this procedure.

TABLE 3

Contrast x-ray methods used in parasagittal meningiomas during various periods

	1924–1933	1934–1946	1947–1954	Total
No. of cases	34	171	75	280
Ventriculograms	22	95	15	132
Encephalograms	2	83	33	118
Angiograms	2	24	64	90

Surgical Procedure. The continuity of the direction of our neurosurgical clinic for more than 30 years necessarily has resulted in a definite continuity of surgical principles and techniques in general, and particularly in the removal of parasagittal meningiomas, probably more than in removal of most other intracranial tumors, a rather systematic sequence of operative steps can be postulated. This continuity is documented by the fact that of the 304 major operations performed on our 280 patients, 212 were carried out by the senior author, 46 by Norlén, 17 by Sjöqvist, and 29 by other members of the staff. (Minor operative procedures, such as re-elevation of bone flaps or removal of infected bone, are of course not included in these figures.)

The surgical principles for removal of parasagittal meningiomas followed in this clinic have been amply discussed in earlier publications. However, some remarks pertinent to the problem of *sinus resections* will follow here.

The relationship between infiltration of the superior sagittal sinus, sinus resection and mortality is presented in Table 4.

TABLE 4

Relation between sinus infiltration, sinus resection and mortality

State of Sinus Type of Resection	1924–46			1947–54			Total		
	No. of Cases	No. of Deaths	Per Cent	No. of Cases	No. of Deaths	Per Cent	No. of Cases	No. of Deaths	Per Cent
Sinus completely obliterated, bloc resection	38	4	10.5%	10	1	10.0%	48	5	10.4%
Sinus partially obliterated, bloc resection	15	3	20.0%	1	0	0.0%	16	3	18.7%
Sinus partially obliterated, marginal resection	47	4	8.5%	33	4	12.1%	80	8	10.0%
	100	11		44	5		144	16	

From the data presented, it appears that from 1947 through 1954 bloc resection of a partially obliterated sinus was carried out in only 1 case, whereas it occurred 15 times between 1924 and 1946. These figures show better than words that the emphasis laid upon a *conservative attitude* towards sinus resections which we expressed years ago has even gained in momentum during the last decade. From Table 4 also the impression may be gained that complete sinus obliteration has been observed somewhat less frequently in recent years. Whether this possibly is a consequence of earlier diagnosis is open to discussion. That a patent sinus should never be resected only for the sake of ease and simplicity of removal of the tumor is, as we think, a point beyond discussion. In our opinion no important cerebral vascular channel should be sacrificed except for very urgent reasons and in our opinion such reasons are rarely present in the case of parasagittal meningiomas.

It is also questionable whether an obliterated sinus should always be resected, especially in cases of parasagittal tumors of the middle and posterior third.

There are some cases in our series in which the patient was made definitely worse by resection of a completely obliterated sinus. The reason for this is that collateral channels may have developed in the falx, which drain the veins of the opposite side, and these veins must be ligated if the sinus is resected in continuity. Resection of the obliterated sinus, especially in the Rolandic area, should therefore not be made unless the operator is satisfied that the drainage of the veins from the opposite hemisphere is not impaired. It might be advisable to abstain from a sinus resection, which might result in a quadriplegic patient, and instead postpone the decision to resect the sinus until a recurrence is present. This may be several years later and meanwhile the collateral circulation may have been further developed so that a sinus resection may be safely carried out.

RESULTS OF OPERATION

Immediate Operative Results. Of the total number of 280 patients, 4 were not operated on. The first 3 of these have been discussed previously;[4] they included 1 patient who died preoperatively from pneumonia, 1 pregnant woman who died after Caesarean section which was to have been followed by craniotomy, and 1 patient in the pre-arteriography era (1932) when the tumor could not be localized. The fourth case (508/47) was that of a woman of 58 years, with a long history of Jacksonian seizures, hemiplegia and typical

TABLE 5

Results of operations

Type of Operation	1924–46			1947–54			Total		
	No. of Cases	No. of Deaths	Per Cent	No. of Cases	No. of Deaths	Per Cent	No. of Cases	No. of Deaths	Per Cent
Exploration	1	1	100.0%	0	0	0.0%	1	1	100.0%
Incomplete removal*	3	1	33.3%	3	0	0.0%	6	1	16.6%
Complete removal	197	23	11.1%	69	8	11.6%	266	31	11.6%
Operation for recurrent tumor (first op. elsewhere)	1	1	100.0%	2	0	0.0%	3	1	33.3%
	202	26	13.0%	74	8	10.8%	276	34	12.3%

* Including 1 case in which the tumor was completely removed in a second operation a few months later (573/53).

x-ray changes who died of pneumonia before operation was carried out. At autopsy a large parietal parasagittal meningioma was found.

The remaining 276 patients were operated on. A total of 304 major operations were performed, including operations for recurrent tumor. The types of operations and results are listed in Table 5. (In this table the re-operations for recurrence of tumor are not included.)

Among the group of *complete removal*, a total of 266 cases, are included a few cases in which removal was completed in several stages. This occurred mainly in some of the earlier cases before 1930, but also in 1 case since 1947 (Case 458/49). From the x-ray studies a left parietal tumor was suspected. At operation only encephalomalacia was found. Reinterpretation of the pictures led to re-exploration 3 weeks later at which time two parasagittal meningiomas in the occipital area were found and removed.

The group of *incomplete removal* includes 6 cases. These cases are discussed in detail below.

Operations on Patients Previously Operated upon Elsewhere. Three of the patients had undergone surgery previously in other hospitals and were re-operated upon in our clinic. One of them died from meningitis after success-

ful removal of the recurrent tumor. The second had a huge recurrence of 150 grams on both sides of the falx; this was successfully removed and the patient was reported well 3 years after the second operation. The third patient had a large meningioma, probably originating from the anterior third of the falx, removed in another clinic in 1952. In 1954 he was reoperated upon here and a huge meningioma filling almost the entire anterior fossa on the left (370 grams) was removed. Invasion of the ethmoidal cells made a radical extirpation impossible. The patient received 10 liters of blood during this procedure. He was discharged 5 weeks later. His present condition is unknown.

Postoperative Mortality. All patients who died in the hospital after surg-

TABLE 6

Causes of postoperative death

Diagnosis	1924–46	1947–54	Total
Exsanguination	6	0	6
Cerebral edema	5	5	10
Tumor not removed; postop. increase of pressure	1	0	1
Infection	3	0	3
Status epilepticus	4	0	4
Pneumonia	3	1	4
Pulmonary embolism	1	1	2
Cardiac insufficiency	1	0	1
Thrombosis of carotid artery	1	1	2
Postoperative clot	1	0	1
	26	8	34

ery, regardless of the time interval between operation and death and regardless of immediate cause of death, are considered postoperative mortalities. There were 34 mortalities, listed in Table 6. Postoperative deaths after second or third operation for recurrent tumor are not included here. The figures up to 1947 are given separately.

It is interesting to note that up to 1947, loss of blood was the most important cause of death. Since then cerebral edema has taken its place. It appears that modern methods of blood replacement during or after surgery have greatly eliminated the dangers of operative blood loss. On the other hand cerebral edema obviously is still a major unsolved neurosurgical problem. In some of the cases the brain swelling was probably caused by prolonged exposure and manipulation of the cerebral tissue during surgery. In 2 cases the edema was already noticeable during the course of the operation. In 1 of these a very large tumor was present making removal difficult; in the other a constriction of numerous cortical veins by the dural edge, with acute hemorrhagic infarction, occurred during removal of a deep meningioma. In 1 case, the postoperative edema probably was caused by the clip-

TABLE 7

Relation of postoperative mortality to location of tumor

Location of Tumor	No. of Cases	No. of Deaths	Per Cent	1924–46 Per Cent
Sinus longitudinalis superior				
Anterior third	56	3	5.4%	6.5%
Middle third	109	14	12.8%	11.0%
Posterior third	31	3	9.7%	8.0%
Falx				
Anterior third	35	7	20.0%	20.7%
Middle third	30	4	13.3%	15.0%
Posterior third	15	3	20.0%	25.0%
	276	34		

ping of both pericallosal arteries during the resection of a meningioma of the lower anterior border of the falx.

The relation of *mortality to tumor localization* is illustrated in Table 7.

The fatalities in cases of meningiomas of the falx have decreased in the last decade whereas they have increased after removal of meningiomas of the middle third of the sinus, possibly ascribable only to statistical variation.

In Table 4 the mortality in relation to *sinus resection* is shown. As stated before in 1947,[4] these figures are too small for definite conclusions. Bloc resection of a not completely occluded sinus was performed only once since 1946, and in this case only the frontal third was involved. The patient made an uneventful recovery.

The mortality in *five-year periods* is presented in Table 8.

We have no ready explanation for the variation in the percentage of deaths during various periods, relatively high from 1947 through 1951, obviously low in the period of 1942–46, and lowest in the last 3 years (3.5 per cent).

Late Results. There were 6 cases in which the removal of the tumor was *incomplete*. One of these patients died immediately after operation.

TABLE 8

Mortality in 5-year periods

Time	No. of Cases	No. of Deaths	Per Cent
1924–31	16	3	19.0%
1932–36	69	9	13.0%
1937–41	57	11	19.3%
1942–46	60	3	5.0%
1947–51	45	7	15.5%
1952–54 (3 years)	29	1	3.5%
	276	34	12.3%

The second patient died 9 months later from progression of the tumor.

In the third patient, a girl of 16 years, a meningioma en plaque was found in the middle third of the sinus with involvement of the overlying bone. Because of the patent and still functioning sinus, no attempts to completely extirpate the tumor were made and only the overlying involved bone with some tumor was removed (bone and tumor weighing 400 grams). One year later this girl had only slight peroneal weakness and episodes of twitching in one foot. She pursued her studies in England and according to the latest report, 9 years after surgery (1954), she is well and working.

In the fourth patient, a man of 56 years, a large meningioma in the right parietal area with extensive infiltration of the sinus but without obliteration was found. To preserve the function of the sinus, and to avoid an extensive resection of sinus and falx, some tumor was left in place. The patient recovered well, but 2 years later focal seizures developed. At that time a re-

TABLE 9

Condition of patients, surviving complete removal of tumor, 1 year after surgery

Dead after discharge but within 12 months after operation	6
Well and working	127
Partially well	52
Invalids	30
Unknown	7
	222

current tumor, the size of a tangerine, was removed which also invaded the tentorium. The sinus was resected as far back as the confluens sinuum. Postoperatively he had an almost complete tetraplegia, and died 2 years after the second operation, probably of recurrent tumor.

The fifth patient, a woman of 45, had a meningioma of the posterior third of the falx with invasion of the sinus rectus and confluens sinuum. It appeared preferable at operation to preserve the still functioning sinus rectus and confluens sinuum and to risk the possibility of a recurrence which then could be removed at a later date after a collateral circulation would have been established. However this patient is reported to be well and working in 1954, 3 years after operation.

The sixth patient, a woman of 51, had a large parietotemporal tumor demonstrated by arteriograms. At surgery, however, a large meningioma of the falx was present in the posterior third, situated quite deep and attached to the sinus rectus. In view of the unsuitable position of the patient, only the right half of the tumor was removed; the remainder was removed successfully 6 months later. The last report, 1 year after surgery, states that she was committed to a mental hospital because of restlessness and probably psychosis.

Among the group of patients with *complete removal* of the tumor, 235 out

TABLE 10

Fate of all patients operated upon, 1 year after surgery

Condition of Patient	No. of Cases	Per Cent
Dead postoperatively or within 1 year after operation	41	14.8%
Well and working	129	47.0%
Partially well	54	19.5%
Invalid	31	11.2%
Unknown	7	2.5%
Fate still undecided (operated on in 1954)	14	5.0%
	276	

of 266 survived. Of these 69 were considered well on leaving the hospital, 124 were improved and 42 were unimproved or worse than on admission.

We feel that the condition of the patient *1 year after discharge* from the hospital is a quite useful means of evaluation of the results of surgery. From this group we have to exclude 13 patients who were operated upon during the last year (1954). There remain 222 in whom removal was complete and successful. The fate of these patients after 1 year is tabulated in Table 9, and the fate 1 year after surgery of *all* patients operated on, is shown in Table 10.

The condition of those patients who have been observed for *5 years* and those who have been followed for *10 years*, is summarized in Table 11.

It is obvious that such late figures are of quite limited value, since in

TABLE 11

Condition of patients operated upon, 5 years and 10 years after operation

Condition of Patient	No. of Patients	Per Cent
Five-year follow-up: 234 cases		
Well and working	58	25.0%
Partially well	70	30.0%
Invalid	25	10.7%
Unknown	25	10.7%
Dead	56	24.0%
	234	
Ten-year follow-up: 176 cases		
Well and working	37	21.0%
Partially well	33	18.7%
Invalid	12	6.8%
Unknown	34	19.2%
Dead	60	34.0%
	176	

TABLE 12

Survival periods of patients with complete tumor removal, who died after discharge from hospital

Survival Time (Years)	No. of Patients
½–1	6
1–2	2
2–4	7
4–6	4
6–8	2
8–10	5
10–15	10
over 15	4
	40

most cases the cause of death is unknown (most of the patients in the 10-year follow-up group are in the age group of 55 to 65), and since we have no report at all on a large number of patients. Of the 222 patients surviving after complete removal, 40 are known to be dead at the time of this report. The interval between discharge from the hospital and death of these patients is presented in Table 12.

We have excluded from this group those patients who were re-admitted and reoperated upon a second or third time for recurrences. How many of these 40 patients died of recurrence is not known, since in almost none of the cases was an autopsy performed.

Frequency of Recurrence. Sixteen of our patients were readmitted and reoperated on for recurrence. The time interval between their first and second operation and between the second and the third may be found in Table 13.

TABLE 13

Recurrent tumors
Time interval between first and second (and third) operation

Time Interval	Between 1st and 2nd Op. (No. of Patients)	Between 2nd and 3rd Op. (No. of Patients)
1 year	1	2
1– 2 years	2	1
2– 3 years	1	
3– 4 years	5	
5–10 years	5	1
over 10 years	2	
	16	4

All of these patients were re-explored, with 1 postoperative death. Of the remaining 15, 6 died later (9, 6, 5 and 2 years, and 2 less than 1 year after their second operation). Four were readmitted and operated on a third time (1, 1, 2, and 6 years respectively after the first operation for recurrence). Of these 4, 1 died postoperatively of meningitis, the other 3 within 1 or 2 years later. Of the 5 remaining patients in this group, 2 were reported well 3 years after operation, 2 were reported worse shortly after discharge, and 1 was reported living but suffering from epilepsy 9 years after his last operation.

SUMMARY

1. Of a total of 5250 verified brain tumors observed in a 30-year period in the Serafimerlasarettet, 1004 were meningiomas, and out of these 280, approximately one-fifth, were parasagittal, including those attached to the falx cerebri.

2. Of the 280 patients with parasagittal meningiomas, 276 were operated on. Among these were 34 fatalities (12.3 per cent). The postoperative mortality in the 80 cases of meningiomas of the falx was 17.5 per cent, and in the remaining 196 cases of other parasagittal meningiomas, 10.2 per cent.

3. Complete obliteration of the sinus longitudinalis superior was found in 48 cases (17.1 per cent), and partial obliteration in 96 (34.3 per cent). The mortality after bloc resection of the completely obliterated sinus was 10.4 per cent, after marginal resection of a partially obliterated sinus 10.0 per cent, and after bloc resection of a partially obliterated sinus 18.7 per cent. Since 1947 bloc resection of a still partially patent sinus was carried out in only 1 case (in the anterior third of the sinus). Emphasis is again laid upon a conservative attitude in regard to resection of a still functioning sinus.

4. Among the causes of postoperative death, cerebral edema takes first place (10 cases, or 29.4 per cent). Blood loss, the principal cause of death in earlier years up to 1947, now ranks only second (6 fatalities or 17.7 per cent).

5. One year after operation, 129, or 47 per cent of all patients operated on, were well and working, 41 (14.8 per cent) were dead, 54 (19.5 per cent) were partially well, 31 (11.2 per cent) were invalid, and of 21 (7.5 per cent) the fate was unknown or as yet undecided.

6. Of the 276 patients operated on 234 were observed for 5 years after surgery. Of these 25 per cent were well, 30 per cent partially well, and 24 per cent dead. Of 176 patients followed for 10 years after operation, 21 per cent were well and working, 18.7 per cent partially well, and 34 per cent dead.

7. Seventeen patients were readmitted and operated on a second time, and 4 were operated on a third time for recurrence of tumor.

8. In preoperative diagnosis, arteriography has become increasingly valuable and during the last decade has been used more often than all air studies combined. Separate injections of the internal and external carotid arteries are recommended.

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PERIPHERAL NERVE SURGERY

THE TWO-STAGE OPERATION*

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FROM time to time, defects in major peripheral nerves are encountered that cannot be overcome by any combination of procedures usually employed for primary suture. This is especially true during time of war when large numbers of nerve injuries are concentrated in neurological centers. When primary repair is impossible, the problem becomes one in which either a two-stage lengthening operation (bulb suture or "stretching" procedure) or a nerve graft must be employed. With the exception of those of Professor Seddon[3] and his associates, nerve grafts have not been successful in a significant number of reported cases. If the defect cannot be made up by either a two-stage operation or a nerve graft, the only remaining possibility for improvement in function of the disabled extremity is an orthopedic operation, a tendon transfer or arthrodesis, either of which is inferior to even mediocre neurologic recovery following nerve repair.

In a series of 1713 peripheral nerve operations performed at one neurosurgical center during World War II, we had no successful experience with nerve grafts. We encountered 92 (5.3 per cent) patients in whom repair was attempted and 77 (4.5 per cent) patients in whom the defect was overcome by the use of a two-stage operation. There were 30 patients requiring a two-stage operation upon whom we obtained follow-up data from 6 to 26 months. There were 52 (3 per cent) patients in whom the nerve injury proved to be irreparable, either before or after an attempted two-stage operation, 15 of them following an attempted two-stage repair.

In a previous publication,[2] we catalogued our experience in overcoming large defects by primary suture and described the method that we employ in accomplishing a primary nerve repair. The essentials of the procedure to overcome such defects consist of wide mobilization of the proximal and distal segments and the use of prolonged fixation of the joints in a position to lessen the nerve defect. The surgeon must be ever cognizant of the fact that every effort should be made at the original operation to do a primary suture without resorting to the two-stage procedure, because the results are never as good in comparable cases when the two-stage procedure is used in contrast

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to a one-stage repair. The tension that is employed in the so-called "stretching operation" unquestionably produces further intraneural damage through scarring and thereby lessens the final recovery of both sensory and motor function. Nevertheless, by whatever means a nerve suture is accomplished, if there is functional recovery, the final result is considered superior to an orthopedic procedure to overcome the loss of function from the paralyzed nerve.

The two-stage operation (bulb suture or "stretching operation") has been employed often in the repair of large defects and the gain in length resulting from it has been attributed to an actual stretching of the nerve. We doubt that it is possible to stretch a major nerve to any significant degree—certainly not over 1 cm. or at the most 2 cm. Our feeling is that the apparent gain in length following traction is ascribable to gradual shortening of the normal course of the nerve. Properly performed traction may provide an additional 5 or 6 cm. in length, but at the same time causes additional injury throughout the entire length of nerve subjected to it. Thirty to 40 per cent of function is lost as a direct result of this traction in contrast to what one might expect had the nerve ends been united without tension and by the use of other procedures to shorten its course. It is for this reason that we consider the two-stage operation a last resort for nerve repair, and preferable only to a nerve graft.

In the literature on peripheral nerve surgery, little attention has been directed toward the method of performing the two-stage operation. It is our belief that damage to the nerve can be reduced to a minimum by observation of certain technicalities which we have employed. Usually, the suture is performed with little regard for the additional amount of injury that results from uniting the proximal and distal lesions to each other with catgut, heavy silk, or wire sutures. Others have sutured the neuromas to fascia, muscle, or periosteum, markedly flexed the joints, and later employed rapid extension within 10 to 14 days during which time the nerve presumably is being "stretched." Actually the nerve is being forcibly pulled from its normally somewhat tortuous course in the extremity. We are certain that this method produces such extensive injury within the nerve, both in the proximal and distal segments, that the already large defect may be actually increased rather than decreased, making eventual repair impossible. When this rapid method of traction and extension is employed, one will find as thin slices of the segments are made in search of an anatomical design somewhat like normal nerve, that progress may extend many centimeters into the proximal or distal ends before anything resembling normal architecture is encountered. The homogeneous mass of fibrosed and degenerated nerve that has destroyed the normal architecture represents damage from the rapid traction applied to the nerve under tension.

After observing several patients with extensive defects from their original wound in whom bulb suture had been done previously, we abandoned the usual technique in favor of one using the same care as that used in a patient undergoing primary repair. Even with this superior method, some

injury to the nerve from traction always occurs near the suture site and as far as 1 or 2 cm. both in the proximal and distal segments, thereby sacrificing a considerable amount in final result. For this reason, we strongly advocate extensive mobilization and the use of every reasonable maneuver to obtain a primary one-stage repair, reserving the two-stage procedure only as a last resort.

METHOD

We believe the following method the least injurious to the nerve, and, if carefully performed, it minimizes fibrosis in the segments adjacent to the proximal neuroma and distal glioma thereby affording a chance of performing definitive suture of reasonably normal nerve ends.

First Stage. As shown in Fig. 1, the nerve both proximal and distal to the lesion is first mobilized through a long incision (a) which will overcome a great deal of the

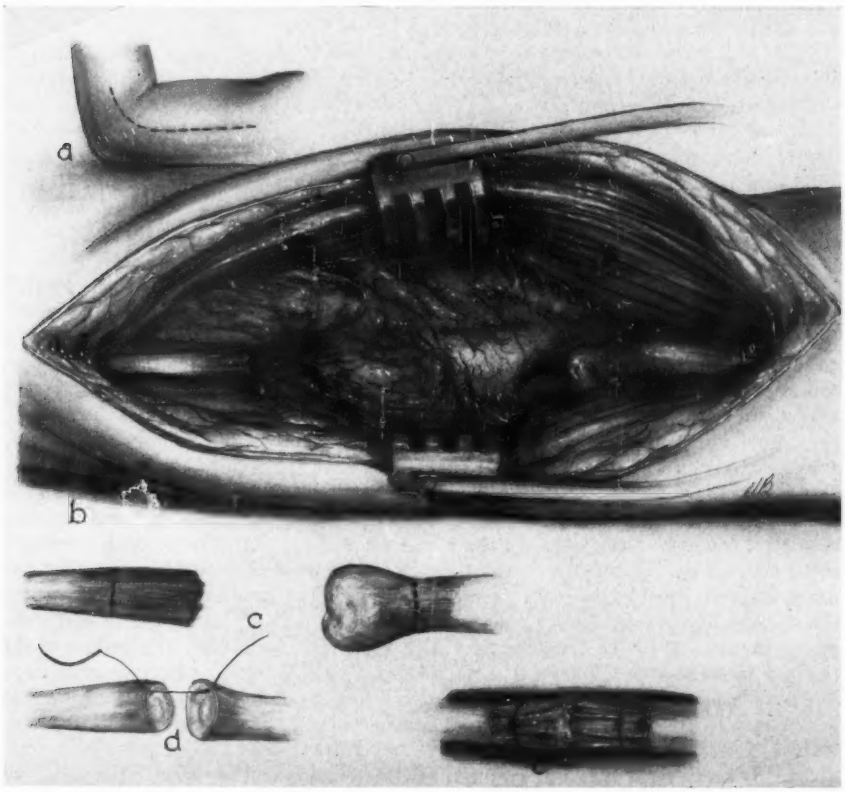

FIG. 1. (a) Outline of skin incision. (b) Lesion exposed and nerve segments mobilized. (c) Line of partial resection of neuromas. (d) Suture of partially removed neuromas. (e) Tantalum foil cuff in place.

apparent defect. This is followed by freeing the severed ends and estimating the size of the defect (b). If it is obvious that the ends cannot be approximated when the neuromas are resected, the proximal and distal segments are mobilized as completely as possible and the two neuromas brought as near together as possible. The defect is further reduced by placing the limb in the position designed to lessen the gap in the nerve. Next, the length of the proximal and distal neuromas is measured to determine what portion can be resected and still accomplish approximation of the ends of the partially removed lesions (c). When this resectable length is determined, excision of this amount of the proximal neuroma and distal glioma is made (d) so that end-to-end suture can be accomplished even if the suture site is under slight tension. In other words, the same method is used for suture of the partially removed lesions as is used in primary suture. For this we use .003 or .005 tantalum wire swedged on a cutting needle and we never employ a sling or retention suture of any sort. The suture site is then wrapped in a cuff of tantalum foil, 3 to 5 cm. long, which is held in place by loosely tied ligatures of plain catgut (e). This is the one instance in which we feel that tantalum foil has a place in peripheral nerve surgery. The wound is closed in the usual fashion and the limb is completely encased in a plaster cast rather than any type of splint or mold.

The cast is left in place 4 to 6 weeks in much the same manner as is done in primary nerve suture, when the defect has been repaired satisfactorily in the original procedure. When the cast is removed, the limb is slowly extended over an additional period of 3 to 4 weeks and under no circumstance is rapid extension permitted. Fortunately, when an extremity is maintained in a cast for this period of time, rapid extension is partially prohibited by moderate fibrosis that occurs in the incorporated joints, and, in addition, the patient is warned against rapid extension of these joints. The 6- to 9-week period between operative procedures will have no appreciable bearing on the final neurologic result.

Second Stage. When full extension of the limb has been accomplished, the second-stage procedure is begun (Fig. 2) by exposing the nerve through the original incision (a) and removing the tantalum foil cuff. During this period, the cuff will lessen the amount of fibrosis that might occur between the suture site in the nerve and the surrounding scar tissue. A thin layer of mesothelial tissue surrounds the nerve in the region where the foil has been applied and a similar layer forms between the foil and the tissues in the nerve bed. The site of suture and the nerve proximally and distally are palpated to estimate the amount of scarring that has resulted from the traction. Investigation by palpation affords a very excellent estimate of the point where the devastating type of scarring is present. This portion of the nerve will feel firm and cord-like, whereas that portion of the nerve where the elements approach normal is soft and pliable. The joints of the extremity are then flexed to be certain that the nerve ends can be approximated when the extensively scarred segment has been resected. Ideally, one would want to resect all of the portion that feels firm and obviously scarred, but it is just as obvious that one cannot excise all of this portion of the nerve if it makes approximation of the ends impossible. Occasionally in such case, one may have to be satisfied in performing a repair involving a portion of nerve that is somewhat distorted by scar tissue. The resection of the involved portion is continued by serially excising small segments in an effort to demonstrate grossly normal-appearing nerve structure. The majority of the resection will be made in the proximal segment. Completely normal nerve as seen on cross section will never be encountered (b and c). When the best possible cross section of nerve is obtained in which suture can be accomplished without undue tension, the nerve ends are approximated using

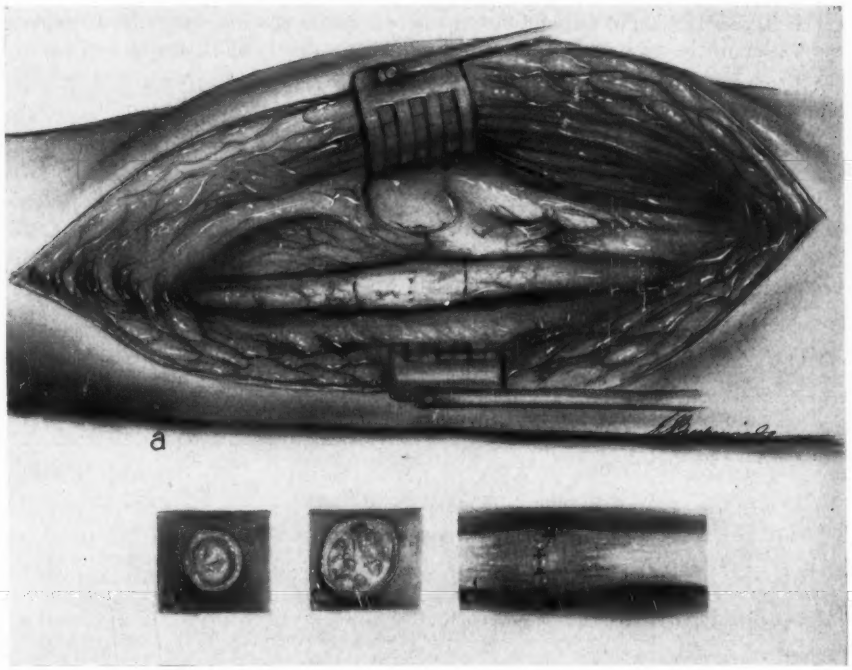

FIG. 2. (a) Exposure of the first-stage suture line and removal of tantalum foil cuff. Line of resection of sutured segments is shown. (b) Gross appearance of distal cross section. (c) Gross appearance of proximal cross section. (d) Second-stage suture completed.

a .003 tantalum wire without a retension or sling suture and without the use of a tantalum cuff (d). The limb is again encased in a circular plaster cast which is maintained for 4 to 6 weeks following which full extension of the limb is gradually allowed over a period of an additional 2 to 4 weeks. When this point has been

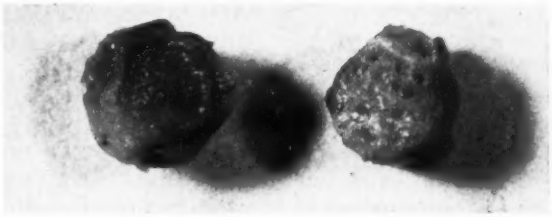

FIG. 3. Median nerve suture, second stage, proximal $\frac{1}{3}$ of arm. (*Right*) Proximal cross section showing abnormal, large nerve fibers and fibrosis. (*Left*) Distal cross section showing degeneration and glistening, homogeneous surface.

First-stage defect 8.0 cm.; second-stage defect 6.0 cm.

Result in 11 months was 60 per cent (fair) with return of crude sensation and sweating and 50 per cent motor power in pronator teres, flexor digitorum sublimis and flexor digitorum profundus. (Mag. ×3)

reached, the position of the wires at the suture site is checked by x-ray. If there is separation of the wire sutures, the suture line has not held and must be redone.

We wish to point out again that we have seen recovery occur following the above-described procedure even though the nerve ends that were sutured appeared grossly nothing more than a homogeneous mass replacing the normal anatomy that one sees in the usual nerve repair (Fig. 3). Admittedly, the final neurologic recovery is small in such case, but it is preferable to complete loss of nerve function. Any return of sensation in an anesthetic area is useful and any motor recovery is preferable to complete loss of nerve function in that muscle. When functional recovery is inadequate,

TABLE 1

Summary of 92 two-stage operations for extensive peripheral nerve defects

Nerve	No. of Operations	Completed	Irreparable Defect	Average Gap			Recovery	
				1st Op.	2nd Op.	1st & 2nd Op.	0–60%	60–80%
Radial	21	15	6	4.2	4.1	8.3	2	0
Median	9	8	1	5.8	5.4	11.3	2	2
Ulnar	17	15	2	6.1	3.7	9.8	5	4
Median and Ulnar	6	6	0	6.5 6.4	5.0 5.6	10.4 11.4	0	2
Sciatic	15	13	2	6.8	5.9	12.9	0	3
Common Peroneal	10	6	4	5.3	6.4	10.1	5	0
Tibial	13	13	0	6.4	4.3	10.6	3	2
Brachial Plexus	1	1	0					
Total	92	77	15	5.9	5.0	10.6	17	13

orthopedic procedures may be required which can always be done at a later date if time proves that they are indicated.

Table 1 shows data on 92 attempted two-stage operations. Of these, 77 were completed and in 15 instances the defects were irreparable. Unfortunately, the follow-up period of 45 patients was inadequate because of their separation from Army service too soon following their second-stage operation. In 30 cases the follow-up observation varied from 6 to 26 months, averaging 11.3 months. Only 2 complete failures occurred following successful two-stage repair: 1 radial and 1 ulnar nerve. Our average result was only fair (55.6 per cent) compared to results of 80–85 per cent obtained in a simi-

lar but larger group of primary nerve repairs.[1] Of the 30 patients with adequate follow-up, 17 showed results up to 60 per cent and only 13 better than 60 per cent, with only one ulnar nerve yielding a result as high as 80 per cent. These data bear out the previous statement, that when a two-stage operation is required, one is penalized in the order of 30 to 40 per cent possible recovery of function.

CONCLUSIONS

1. A method of performing a two-stage operation in cases of extensive peripheral nerve defects which will result in minimal traction injury to the adjacent and normal nerve substance is described and illustrated.

2. The two-stage operation is a last-resort procedure and should be used only in preference to a nerve graft when it has become obvious that primary suture cannot be employed. We believe this procedure is preferable to nerve grafting.

3. Results obtained by this method are compared with those of primary suture.

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CEREBRAL ARTERIAL SHUNT IN THE MONKEY*

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THE purpose of this paper is to present observations which suggest that the tonus of extracranial arteries of the head may be a significant factor in determining blood flow to the brain. It is well known that the pressoceptive reflexes are of fundamental importance in regulating the cerebral blood flow. One of the most important aspects of this regulation concerns the effect of pressoceptive reflexes upon the tonus of cephalic blood vessels.

The main purpose of the pressoceptive reflexes appears to be the maintenance of a more or less constant blood flow to the brain. This purpose is achieved, at least in part, by widespread vasomotor regulation of peripheral arteries including extracranial vessels of the head. On the other hand, brain arteries are not thus involved to a significant degree.[1,5]

Heymans, noting the difference in vasomotor reactivity of extracephalic arteries of the head as compared to brain arteries, concluded that this difference was of functional importance. He stated, "The extracranial tissues, mainly the vessels of the face, in reality function as shunts and blood reservoirs for the cerebral circulation to protect the brain against variations in blood pressure and to regulate the cerebral blood flow."[4]

Heymans' theory was based upon experiments in dogs. His hypothesis has received support from studies in the cat.[2,7] However, both of these animals possess a rete mirabile which affords a rich anastomosis between the external carotid artery and the circle of Willis.

It has been generally assumed that an arterial shunt in the cranial circulation could exist only in animals that possess a rete mirable. That such an assumption may be erroneous is suggested by the results of the following experiments in the rhesus monkey, whose cranial circulation is comparable to that of man as regards communications between the intracranial and the extracranial circulations. The present study lends support to the concept that the intracranial and extracranial circulations of the head are functionally interrelated even in the absence of a rete mirabile.

METHOD

Carotid arteriography under light pentobarbital anesthesia was employed as the method for investigating the effects of stimulation of the cervical sympathetic trunk

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upon the cephalic circulation. Thorotrast (20 per cent thorium dioxide) was the contrast medium. It was delivered to the common carotid artery ipsilateral to the stimulated cervical sympathetic trunk by a controlled pressure injection system at a pressure of 750 mm. Hg. The injection, always made ipsilateral to the stimulation, coincided exactly with the 2-sec. exposure of the roentgenogram and usually amounted to 3 cc. of Thorotrast. Other roentgenographic factors were as follows: 65 kV., 15 mA., cardboard cassettes, tube-film distance 48 cm. A thyratron-type stimulator which emitted a saw-tooth wave form of 4-msec. duration was used. Parameters of stimulation were 2.5–5 V., 30–120 sec., and 60 c./sec. except for one stimulation of 180 c./sec. The effectiveness of the stimulation was verified in each instance by noting the pupillary response. Three control arteriograms were made at 3- to 5-min. intervals, the stimulation was accomplished, and several post-stimulation arteriograms were obtained. In one experiment arteriograms were made before and after ipsilateral division of the cervical sympathetic nerve.

RESULTS

Arteriograms satisfactory for interpretation were obtained in 10 experiments in which 14 unilateral stimulations of the cervical sympathetic nerve were carried out (Table 1). The usual effect of the stimulation was to cause a marked constriction of the ipsilateral extracranial branches of the common carotid artery. Constriction of the ipsilateral internal carotid, middle cerebral and first portion of the anterior cerebral artery occurred less constantly and to a lesser degree. The above vasomotor changes were usually associated with (1) an apparent shunting of the injected Thorotrast from the extracranial circulation to the intracranial circulation at the bifurcation

TABLE 1

Monkey	Stimulation	Constriction				Arterial Shunt to Contralateral Side	Arterial Shunt to Brain Stem
		Common Carotid	External Carotid	Internal Carotid	Middle Cerebral		
#31	4 V, 60 cps, 1′	No	Marked	Slight	No	Yes	Slight
#56	4V, 60 cps, 2′	No	Marked	Slight	No	Yes	Yes
	4V, 60 cps, 2′	No	Marked	No	No	Yes	Yes
#30	3V, 60 cps, 1′	No	Marked	Marked	Marked	No	No
#27	2.5 V, 60 cps, 1′	No	Marked	Questionable	Questionable	Yes	Yes
#29	3V, 60 cps, 30″	Moderate	Marked	Marked	Marked	Yes	Yes
#34	3V, 60 cps, 1′	No	Slight	Moderate	Moderate	Questionable	Questionable
#25	2.5 V, 180 cps, 2′	No	Slight	Moderate	Moderate	Yes	Yes
H#11	3V, 60 cps, 2′	Slight	Moderate	Artery visualized	No	No	No
	5V, 60 cps, 2′	Marked	Moderate	Artery visualized	No	Questionable	No
#55	4V, 60 cps, 2′	Minimal	Slight	Artery visualized	No	No	No
	4V, 60 cps, 3′	No	Marked	Artery visualized	No	Yes	Yes
#23	4V, 60 cps, 2′	No	Marked	Artery visualized	Not visualized	No	No
	4V, 60 cps, 2′	No	Marked	Artery visualized	Artery visualized	Yes	Slight

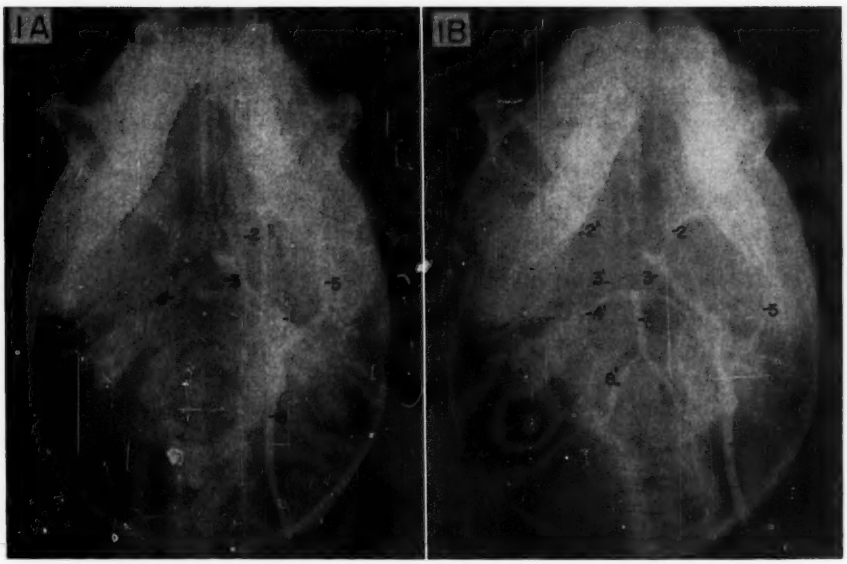

FIG. 1. *Monkey #27.* (A) Control, pre-stimulation arteriogram in the vertex-submental projection. Intracranial arteries visualized are the internal carotid (1), middle cerebral (2), posterior communicating (3), and posterior cerebral (4) arteries ipsilateral to the injection. The posterior cerebral artery of the contralateral side is also visible (4′).

(B) Arteriogram made immediately following a 2.5 V., 60 c., 60 sec. stimulation of the cervical sympathetic trunk ipsilateral to the injection. Severe constriction of the external carotid artery (5) and other extracranial arteries is evident. The apparent intracranial shunt of Thorotrast injected into the common carotid artery (6) is evidenced by visualization of the basilar artery (7), both vertebral arteries (8), and the contralateral posterior communicating (3′) as well as the contralateral middle cerebral artery (2′).

of the common carotid artery and (2) a shunting of the contrast medium to the brain stem and to the opposite cerebral hemisphere by way of the circle of Willis (Figs. 1, 2 and 3). Similar alterations in cephalic blood flow were not observed in any of the control arteriograms.

In only one experiment was the effect of dividing the sympathetic trunk studied. In this experiment the trunk section was carried out 12 minutes following its stimulation. An arteriogram made 5 minutes subsequent to the section revealed that the intracranial arteries remained constricted as compared to control arteriograms made before the stimulation.

Arteriographic evidence that the large arteries at the base of the brain respond to stimulation of the cervical sympathetic nerve was obtained. Post-stimulatory constriction of the internal carotid artery was seen after 6 of the 14 stimulations, the ipsilateral middle cerebral artery constricted after 4 of the stimulations, and the first portion of the anterior cerebral artery constricted after 2 stimulations. The constriction of intracranial arteries at the base of the brain caused by stimulation of the cervical sympathetic nerve was present within 30 seconds of the onset of the stimulation and was observed

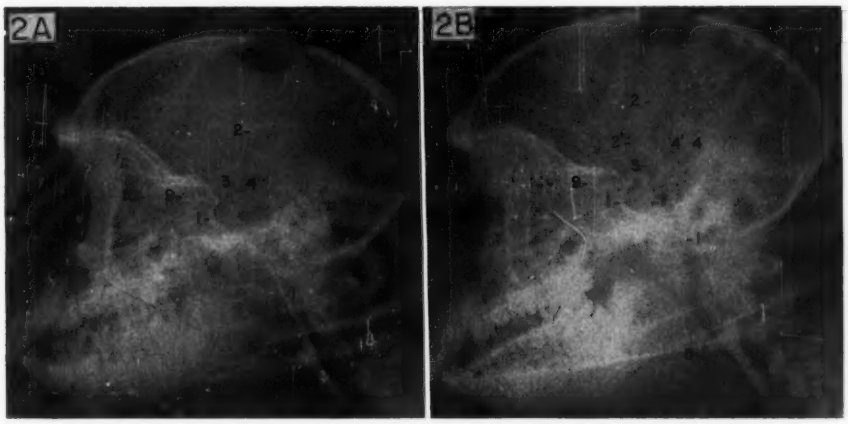

FIG. 2. (A) Control, pre-stimulation arteriogram, lateral view. Arteries visible on the injected side are: common carotid (6), external carotid (5), internal carotid (1), ophthalmic (9), retinal plexus (10), anterior cerebral (11), middle cerebral (2), posterior communicating (3), posterior cerebral (4).

(B) Arteriogram made at the close of a 4 V., 60 c., 2 min. stimulation of the cervical sympathetic trunk ipsilateral to the injection of Thorotrast. The severe constriction of extracranial arteries is evident. Note the almost complete absence of filling of the external carotid artery (5). An intracranial shunting of the contrast medium is indicated by the bilateral filling of the middle cerebral (2, 2') and posterior cerebral (4, 4') arteries. The diversion of Thorotrast through the posterior communicating artery (3) to the basilar artery (7) is noted. The double shadow of the retinal plexuses (10) indicates bilateral filling of the ophthalmic artery.

An arteriogram made 4 min. later revealed a persistence of the above shunt but it was becoming less evident. A repeat stimulation gave the same results as described above.

to persist as long as 17 minutes after cessation of the stimulus although it usually subsided after 2 or 3 minutes.

The common carotid artery evidenced moderate constriction after 3 of the 14 stimulations (Fig. 3) but usually the artery appeared to distend in direct proportion to the degree of constriction produced in the external carotid artery (Figs. 1, 2, and 4).

No attempt has been made to measure the diameter of arteries demonstrated on arteriograms or to apply statistical analysis to such values because a similar study in cats[2] indicated that a change in caliber of an artery easily seen on arteriograms was statistically significant whereas a questionable alteration was not. Fig. 3 illustrates the maximal degree of constriction of cephalic arteries observed in these experiments. It is evident that in this instance the internal carotid and middle cerebral arteries constricted to approximately 60 per cent of their pre-stimulation caliber whereas certain extracranial arteries constricted almost to the point of obliteration.

In 3 of the 10 experiments a curious failure to visualize the ipsilateral intracranial arteries was evident in 20 of the total of 21 control arteriograms. The internal carotid artery did not fill in 15 of the 21 pre-stimulation arteriograms. The external carotid system was demonstrated to its peripheral ramifications in every instance. Since these 3 experiments gave important evidence that stimulation of a cervical sympathetic trunk tends to shunt blood

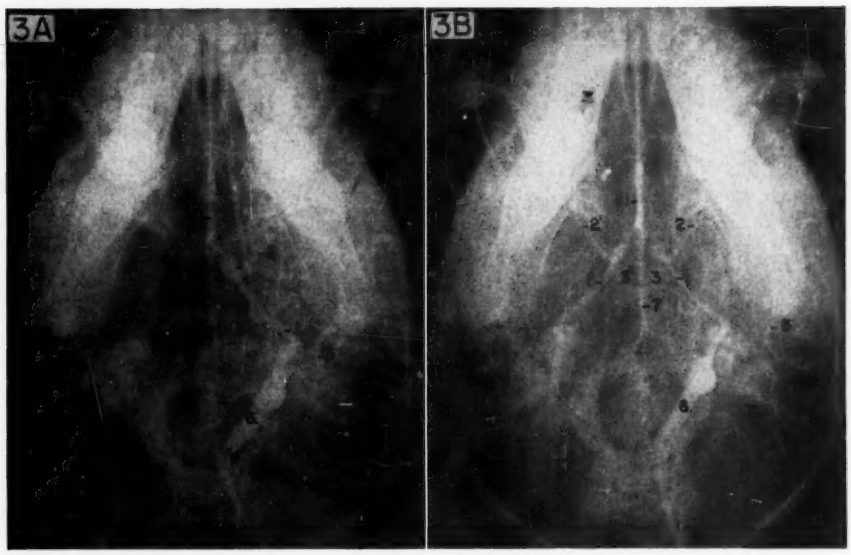

FIG. 3. *Monkey #29.* (A) Control arteriogram made 3 min. before a 3 V., 60 c., 30 sec. stimulation of the cervical sympathetic trunk ipsilateral to the intracarotid injection of Thorotrast. The following arteries are filled: common carotid (6), external carotid artery (5), internal carotid (1), middle cerebral (2) and anterior cerebral (11).

(B) Arteriogram made 2 min. following the stimulation. Extracranial and intracranial arteries ipsilateral to the stimulation are severely constricted. A shunt of Thorotrast to the opposite hemisphere and to the basilar artery (7) is evident.

to the brain from the extracranial portion of the cephalic circulation by reason of constriction of the extracephalic branches of the common carotid artery, they will be reported in some detail.

In the first of these 3 experiments, 7 control arteriograms were made. In none of the controls was even the first part of the internal carotid artery visualized despite the fact that the external carotid artery was filled to its terminal branches (Fig. 4A). Three post-stimulatory arteriograms were obtained. In the first arteriogram, the internal carotid artery filled as far as the carotid syphon only. In the second, the internal carotid and middle cerebral arteries were visualized. In the third post-stimulation arteriogram, excellent filling of brain arteries was obtained which demonstrated the typical shunting of Thorotrast to the contralateral hemisphere and to the brain stem (Fig. 4B).

In the second experiment, there were 8 control arteriograms. The internal carotid artery did not fill in 7 of them and filled only faintly in the remaining arteriogram although in each instance visualization of the external carotid system was excellent. Two post-stimulation arteriograms were made. In one of them the internal carotid artery was well visualized but no intracranial arteries were filled. In the other post-stimulation arteriogram, excellent filling of the internal carotid and brain arteries was

FIG. 4. *Monkey #55.* (A) A control arteriogram failed to fill the internal carotid artery or intracranial arteries despite good filling of the external carotid artery (5).

(B) Arteriogram made immediately after a 3 min., 60 c., 4 V. stimulation of the cervical sympathetic trunk ipsilateral to the injection of Thorotrast. Marked constriction of the external carotid artery (5) is evident. Bilateral filling of the internal carotid (1, 1′) and middle cerebral (2, 2′) arteries occurred. The anterior cerebral (11), posterior communicating (3), posterior cerebral (4) and basilar arteries (7) are also visualized. The intracranial shunt appears to have been caused by constriction of the extracranial branches of the common carotid artery. The shunt to the opposite hemisphere occurred largely by way of the anterior portion of the circle of Willis so that the shunting of Thorotrast to the brain stem was minimal.

seen with the characteristic shunting of Thorotrast to the contralateral hemisphere and to the brain stem.

In the last experiment 6 control arteriograms were made. In the first control arteriogram, excellent filling of the internal carotid and intracranial arteries was obtained. However, none of the remaining control arteriograms demonstrated the internal carotid artery. Two arteriograms made after stimulation of the cervical sympathetic nerve demonstrated the internal carotid artery but not its intracranial branches. The external carotid artery was well visualized to its distal branches in each case.

DISCUSSION

The method of arteriography has allowed simultaneous visualization of the intracranial and the extracranial arterial circulations of the head. This circumstance gave evidence that stimulation of a cervical sympathetic nerve resulted in reapportionment of the cephalic blood flow. In addition, the method allowed indirect measurement of the arteries at the base of the brain and demonstrated that their caliber can be altered by such stimulations.

In the monkey, stimulation of a cervical sympathetic nerve results in severe constriction of extracranial arteries of the head and neck. If, then, Thorotrast is injected into the common carotid artery on the side of the stimulation, most of the contrast medium enters the internal carotid artery. As the Thorotrast enters the circle of Willis it is further diverted to the opposite hemisphere and to the brain stem usually by way of the posterior communicating arteries. The intracranial shunting of Thorotrast seems to be closely associated with constriction of extracephalic branches of the common carotid artery. Further evidence that constriction of extracranial arteries of the head and neck results in diversion of carotid blood flow to the brain was afforded by 3 experiments in which pre-stimulation arteriograms failed to demonstrate intracranial arteries in 20 of the total of 21 control arteriograms. The reason for this curious failure to visualize intracranial arteries was not apparent since the external carotid system was filled well beyond the bifurcation of the common carotid artery. Perhaps spasm of the internal carotid artery occurred. Whatever the explanation, it was of great interest to note that when constriction of extracranial branches of the common carotid artery was produced by stimulating the cervical sympathetic trunk, Thorotrast entered the internal carotid artery in each of the 6 post-stimulation arteriograms made in these 3 experiments and the typical shunt effect was apparent in 2 of the 6 post-stimulation arteriograms.

When Thorotrast was shunted to the brain subsequent to stimulation of the cervical sympathetic nerve, it was also diverted in part to the opposite hemisphere as well as to the basilar artery by way of the circle of Willis. Probably two factors are important in effecting the arterial shunt which occurred at the circle of Willis. Firstly, any sudden influx of blood to the brain by way of one internal carotid artery would be dispersed to all branches of the circle of Willis as a purely passive hydrodynamic function of the circle of Willis. Secondly, the increased vasomotor tone of the middle cerebral and, less apparently, of the anterior cerebral artery tended to shunt blood caudally and to the opposite hemisphere, usually by way of the posterior communicating arteries. Dumke and Schmidt[3] postulated that such a reapportionment of total blood flow within the brain might occur as the result of stimulation of the cervical trunk consequent to a single experiment in a monkey in which they successfully measured separately the blood flow in the internal carotid and the basilar arteries. Carotid arteriographic studies in the cat also demonstrated frequent filling of the basilar and even the vertebral arteries after stimulation of the cervical sympathetic nerve ipsilateral to the injection.[2]

In addition to supporting the concept of a reciprocal relationship existing between the extracranial and the intracranial cephalic circulations, the method of arteriography demonstrated that certain intracranial arteries at the base of the brain have a demonstrable vasomotor innervation. The degree of their vasomotor reactivity was considerably less than for extracranial cephalic arteries of comparable diameter but it was none the less definite. The internal carotid artery was the most reactive intracranial artery, followed by the middle cerebral artery, then by the first portion of

the anterior cerebral artery. No vasomotor effect visible on arteriograms was apparent in the posterior communicating, basilar or vertebral arteries subsequent to stimulation of the cervical sympathetic trunk. The change in caliber of these intracranial arteries was noted only ipsilateral to the stimulation. Since this change was absent in control arteriograms as well as on the side contralateral to the stimulation, there can be little doubt that it represents a true vasomotor innervation of the arteries rather than their response to the contrast medium. Cervical sympathectomy in one experiment had no significant effect upon the caliber of the large intracranial arteries at the base of the brain of the monkey in agreement with results in the cat.[2]

It would appear that the most important implication of the present study is to indicate that the role of the cervical sympathetic nerves in the regulation of the circulation of the brain may be concerned chiefly with their function as a part of the protective mechanism mediated by the pressoceptive reflexes. When the pressoceptive reflexes are elicited by a fall in blood pressure at the carotid sinuses and the aortic arch, the subsequent reflex vasoconstriction of cephalic arteries may be similar to the results obtained by electrical stimulation of the cervical sympathetic nerves since these nerves are the chief efferent pathways of the pressoceptive reflexes to the head. In the monkey, whose cephalic circulation is comparable to that of man in the paucity of arterial communications between the intra- and extracranial circulations, such stimulations appear to initiate a shunt of carotid blood flow to the brain from extracephalic tissues of the head and neck. In addition, a reapportionment of cerebral blood flow to brain areas irrigated by the posterior portion of the circle of Willis, the posterior cerebral, basilar and vertebral arteries seems to occur. Since the method of arteriography does not afford accurate, quantitative data concerning cerebral blood flow, the implications given by this method are in need of further study by other technics such as the use of a radioactive gas as described recently by Kety *et al.*[6]

The possibility that stimulation of the cervical sympathetic nerves or elicitation of the pressoceptive reflexes might result in a differential increase of blood flow to the brain stem is not an illogical consideration. Certainly it would be unexpected to find that the net effect of vasoconstriction of cephalic arteries induced by the pressoceptive reflexes would be a decrease in blood flow to the vital regions of the brain which the reflexes serve to protect.

It would seem from the results of arteriographic studies in the cat and the monkey that the cervical sympathetic nerves affect the cerebral circulation mainly by mediating reflex activity above and beyond the resting state, for stimulation of the nerves causes striking changes in the cephalic circulation as well as constriction of certain large arteries at the base of the brain, whereas section of the nerves results in no demonstrable effect in either regard.

SUMMARY

1. The method of arteriography was utilized to study the effect of unilateral stimulation of the cervical sympathetic trunk upon the cephalic circulation of 10 rhesus monkeys.

2. The usual effect of the stimulation was to cause a marked constriction of extracranial branches of the common carotid artery.

3. Constriction of the ipsilateral internal carotid, middle cerebral and first portion of the anterior cerebral artery occurred less constantly and to a lesser degree.

4. The above vasomotor changes were usually associated with an apparent shunting of the injected Thorotrast from the extracranial cephalic circulation to the intracranial circulation at the bifurcation of the common carotid artery.

5. Within the brain, a shunting of the contrast medium to the brain stem and to the opposite hemisphere by way of the circle of Willis was evident.

6. These experiments support Heymans' theory that a reciprocal relationship may exist between the extracranial and the intracranial components of the cephalic circulation. A rete mirabile in the cephalic circulation is not a necessary component of the cephalic shunt mechanism.

7. The study suggests that the pressoceptive reflexes may protect the brain during a fall in the systemic blood pressure by evoking a shunt of extracranial cephalic blood flow to the brain as the result, at least in part, of the lesser degree of vasomotor reactivity of arteries of the brain as compared to those of the extracranial cephalic circulation.

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CASE REPORTS AND TECHNICAL NOTE

DISSECTING ABSCESS OF THE FALX CEREBRI

CASE REPORT

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(Received for publication July 15, 1955)

The falx cerebri attaches anteriorly at the crista galli in proximity to the cribriform plate and to the frontal and ethmoid sinuses. Two layers of dura mater form the falx during its embryological development, later becoming coherent. In view of the close proximity of the anterior attachment of that structure to potential sources of bacterial infection, it seems fortunate that experience is limited with infection that enters the falx and dissects apart its two layers to form an abscess. No previous case with this complication has been found. It is possible that such a case may have been previously reported as a complication of brain abscess. Reported in that manner, it may not be easily revealed through the usual methods of literary research. The case reported here is the only one in the author's experience. Inquiry of associates indicated that they too had not heard of this entity. Consequently, it is believed worth while to bring it to attention.

It may later become evident that the use of antibiotics is the reason that such an abscess can develop. Previously the patient would have expired from one or more brain abscesses before sufficient time had elapsed for the development of a dissecting abscess of the falx cerebri. No doubt a considerable period of time must elapse for the separation of these two layers of dura mater. It also seems probable that antibiotics do not reach this region as thoroughly as they do the brain in view of the relative avascularity of the region as compared to the brain.

CASE REPORT

The patient was a white male college student, 20 years of age, who was admitted to the Hedgecroft Hospital, Houston, Texas, on Aug. 24, 1952. A history was obtained of headache of 2 weeks' duration. On the day prior to admission he had noted weakness of his right leg. It was thought by the examining physician that his speech was slow.

A spinal puncture was done on Aug. 24, 1952. The pressure was not measured. The cell count was 35 WBC, of which 70 per cent were polymorphonuclear and 30 per cent mononuclear cells. The protein was 25 mg. per cent. A provisional diagnosis of polioencephalitis was made by the admitting neurologist in view of the presence of a local epidemic of poliomyelitis in which cerebral manifestations were not uncommon.

Examination. When he was first seen by the author on Aug. 28, 1952, the findings were as follows: Temperature 101.4°, pulse rate 72, and blood pressure 120/62. The appearance was that of an acutely ill young white male. His neck was quite stiff. Kernig's sign was positive. There was complete motor aphasia. He was apparently able to understand, and he obeyed simple requests well. He would look toward a noise and toward a spoken voice. There was a

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mild divergent squint but no paralysis of extraocular movements. There was no papilledema. He would not attempt to swallow, although it was stated that he did swallow on the day prior to this examination. He was able to squeeze both hands quite well on request. There was no inequality of the strength of the hand grip, although there was generalized weakness accompanying his acute illness. All extremities were appreciably spastic. He was unable to move the right leg at all. There was good strength in the left leg. Tendon reflexes were approximately symmetrical. Babinski response was positive on the right and normal on the left. Abdominal and cremaster responses were absent.

It was believed probable that the patient had a cerebritis on the basis of a bacterial infection. It did not appear likely that he had a localized abscess at that time.

Subsequently a second spinal puncture was done on Aug. 29, 1952, which showed an initial

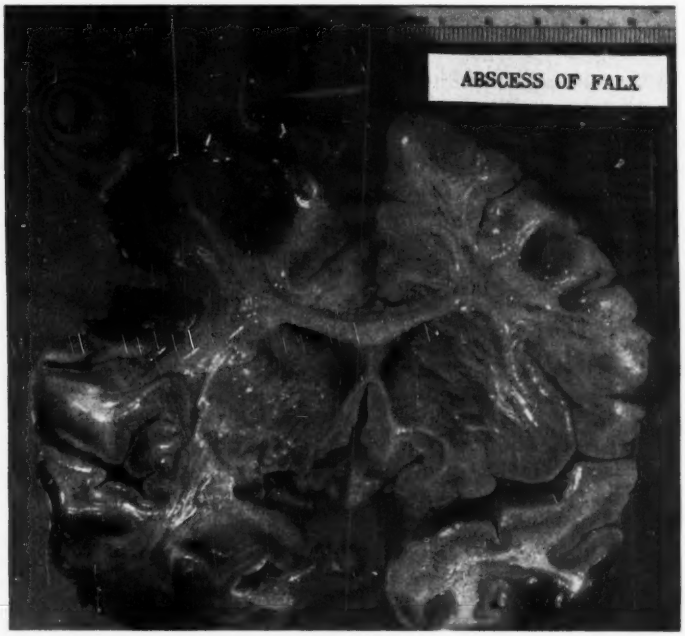

FIG. 1

pressure of 520 mm. of water and a cell count of 400 WBC, of which 94 per cent were polymorphonuclear cells.

Course. Beginning on Aug. 25, 1952, and for 3 weeks he received antibiotics (500,000 units of penicillin IM, q. 2 hr., and 200 mg. of Terramycin IV, q.i.d.). During this period his clinical findings gradually improved. Spinal punctures on Sept. 4, 8, and 22, 1952 showed progressive drop in pressure, the pressures being 450 mm., 320 mm., and 280 mm., respectively. The spinal fluid protein also dropped from 84 mg. per cent to 23 mg. per cent. Pleocytosis diminished to 10 cells, 2 of which were polymorphonuclear. The aphasia cleared satisfactorily. He was free of focal symptoms except for mild weakness in his right leg. However, manifestations of increased intracranial pressure occurred in spite of the improvement in aphasia and paralysis. Bilateral papilledema was noted first on Sept. 1, 1952. The papilledema progressed in spite of his other improvement. On Sept. 27, 1952, after 3 days off of antibiotics, his headaches recurred with vomiting. On Sept. 29, 1952, for the first time since early

in his illness, he again had difficulty in speaking. Paresis of the right leg increased. There was no elevation of temperature at all.

He was subsequently transferred from the Hedgecroft Hospital to the Hermann Hospital. Roentgenograms of the skull showed no calcification in the pineal gland. There was clouding of the right frontal sinus and of the right maxillary sinus as well as the ethmoids with loss of the mucoperiosteal line in the right frontal sinus. Previous films had not shown these changes.

Operation. On the night of Sept. 29, 1952, a left posterior parietal burr hole was made under local anesthesia. After the dura mater had been opened through the burr hole, the brain was found to be under increased pressure. A ventricular needle was inserted into the brain in the direction of the left temporal lobe, since it was felt that an abscess might be present there. The lateral ventricle was never entered. The ventricular needle entered a brain abscess which drained purulent material freely. Approximately 50 cc. of yellowish pus were obtained; and the abscess was irrigated. The ventricular needle was then inserted in other directions. Another smaller abscess was entered and irrigated. During this procedure (under local anesthesia), the patient's condition was fairly good in regard to his state of consciousness.

Course. Soon after return to his room his general condition seemed much worse. He became completely hemiplegic on the right side, and his aphasia was also complete. His respirations rose to 36 and then decreased gradually to 8. With oxygen his color remained good. At 6:30 a.m. on Sept. 30, 1952, a ventricular needle was again inserted through the burr hole and two more separate and distinct brain abscesses, more medial than the previous ones, were tapped and gently irrigated with sterile normal saline. Approximately 2 ounces of pus were obtained from these two brain abscesses. Respirations remained slow. The blood pressure rose. Gradually the respirations failed in spite of stimulation with coramine, and he expired at 7:15 a.m. on Sept. 30, 1952.

Autopsy. There were multiple brain abscesses in the left cerebral hemisphere. A major abscess was present in the left frontal lobe. *The unusual finding was that the two dural layers of the falx had been separated from one another. There was a sac of pus extending from the rostral end of the falx to the caudal end, forming a large expanding lesion between the two cerebral hemispheres* (Fig. 1).

COMMENT

The unusual finding of an abscess of the falx with separation of the two dural layers, both grossly and microscopically, has not been found described in the literature. It is apparent that the patient had meningitis and rather marked cerebritis which was suppressed with antibiotics. After antibiotics were discontinued, the multiple brain abscesses manifested themselves. The abscess within the falx was neurologically silent, the aphasia and paresis of the leg probably arising from the cerebral abscesses. Development of papilledema as the aphasia and paralysis subsided was probably caused by the presence of the abscess in the falx. The development of this unusual abscess is possibly explained by the difference in vascularity between brain and falx with resulting higher concentration of antibiotics in the brain.

At present no means can be conceived whereby this lesion could have been reliably localized and drained by use of standard methods. However, it is possible that arteriography would have shown separation of the anterior cerebral arteries.

SUMMARY

The clinical and autopsy features of a case of multiple brain abscesses of the left cerebral hemisphere with the unusual complication of a very large dissecting abscess of the falx are presented. The possible relationship in the development of this condition between antibiotics and the relative avascularity of the falx as compared to the brain is suggested.

MULTIPLE OCCURRENCES OF GLIOMAS IN A FAMILY

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(Received for publication July 22, 1955)

Intracranial tumors are said to account for over 1 per cent of all deaths.[9] However, the occurrence of gliomas in several members of one family has rarely been recorded. It is our purpose to report the occurrence of 2 verified gliomas and 1 presumed glioma in 2 non-identical siblings and a nephew.

Koch described similar multiplicity in two German families. In one of his families gliomas (glioblastomas) developed in a mother, daughter and son.[4] The Schie family[5] included A. with a proven left temporal astrocytoma. An uncle of A. had a left posterior cerebral tumor without microscopic identification. This patient expired with evidence of intracranial hypertension at the age of 17 years after morbidity of 1 year. Another uncle of A. died at the age of 65 years following a history of tumor with terminal intracranial hypertension. The lesion was presumed to be a right cerebral tumor but was not confirmed.

Riese, Meredith and Zfass[8] reported the development of gliomas in a brother and sister at about the same age (55 years); these tumors were similar histologically. In Leavitt's[6] report of cerebellar tumors occurring in identical twins, one was proven to be a medulloblastoma and the other had caused similar clinical findings. Bing[2] reported the occurrence of cerebral neoplasms in 3 members of a family without other data. Klemme[3] has identified gliomas in 2 members of a family. Astrocytomas in 2 members of a family were treated at the New York Neurological Institute during the experience of one of the authors (A.H.H.).

CASE REPORTS

Case 1. W.L., a 52-year-old white male, was admitted to Johns Hopkins Hospital in July, 1936, under the care of Dr. Walter Dandy. Approximately 2 months previously he had had slurring of speech and for the last 2 weeks he had had right facial numbness. Twitching attacks followed by transient aphasia had occurred. No headaches were noted.

Examination. The patient was right-handed, with right facial hypesthesia and paresis, slurring of speech and absent abdominal and cremasteric reflexes.

Operation. A left craniotomy was performed by Dr. Dandy, whose operative record states: "In Broca's area there was an infiltrating tumor which had produced reddening of the surface of the convolutions and also widening and flattening of them. This region was quite hard. It was in such a position that its removal would have almost certainly caused motor aphasia, although this is sure to come with the progress of the tumor." A small piece of tissue just anterior to Broca's area was removed for diagnosis, and a large temporal decompression was performed.

Course. Death occurred soon after.

Pathological Diagnosis. The surgical specimen contained no neoplastic cells. Although lacking histological verification the tumor was identified as a glioma by Dr. Dandy.

Case 2. E.L., a 64-year-old white male and brother of Case 1, was admitted to the Nix

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Memorial Hospital in San Antonio, Texas, on Sept. 11, 1952. Eight weeks previously he had suffered a sudden coma of 6 hours' duration with residual, rapidly receding, hemiparesis. After a 4-week interval of relatively good health there was onset of progressive aphasia, confusion, vomiting and headache.

Examination. He was a right-handed man who was overreacting and jocose. He had a right hemiparesis, sensory aphasia, astereognosis of the right hand and blurred disc margins.

Roentgenograms of the skull were normal. Electroencephalograms discovered a left temporoparietal focus. Initial pressure on lumbar puncture was 296 mm.; the fluid contained 61 mg. per cent total protein.

Course. The patient refused a craniotomy and expired on Oct. 13, 1952.

Autopsy. A large infiltrating left frontal tumor, 6.5×6 cm. in size, was encountered. Dr. J. W. Kernohan's histological diagnosis was Grade III astrocytoma (Figs. 1 and 2).

Case 3. A.L., a 46-year-old paternally related nephew of Cases 1 and 2, entered a hospital in Phoenix, Arizona in October, 1950, under the care of Dr. John Green. He presented a 3-month history of personality change, headaches and left-sided paresthesias.

Examination. The patient was right-handed, with definite evidence of increased intracranial pressure, left astereognosis and dysesthesias.

Spinal fluid pressure and protein were elevated. Ventriculography suggested a right intraventricular tumor.

Operation. At craniotomy a right paraventricular tumor was encountered and grossly removed.

Course. The patient expired after 2 months' hospitalization.

Histological Diagnosis. The tumor was a malignant glioma varying from a protoplasmic astrocytoma to a glioblastoma multiforme in character (Figs. 3 and 4).

DISCUSSION

Though the nervous system is host to many hereditofamilial disease processes, it is generally held that no genetic influence on gliomas exists in the human family.[1,7] The familial nature of various degenerative diseases and certain neoplastic involvements is well recognized. Generalized neurofibromatosis, multiple enchondromata, von Hippel-Lindau disease, and glioma (more properly neuroepithelioma) of the retina show hereditary influence. Bailey[1] (page 6) made the statement, "No glioma of the brain has even been supposed to be hereditary. . . ."

Brain tumors are common, with figures showing that they make up to 1.8 per cent of all tumors[1] and account for 1 per cent of all deaths.[9] Gliomas comprise some 40 per cent of brain tumors. With such a moderately common affliction multiple occurrences of gliomas among members of the same family should appear occasionally by coincidence. However, the occurrence of 2 proven gliomas and 1 presumed glioma in two generations of the family reported herein is a distinctly unusual coincidence. The generation in which 2 gliomas occurred included a total of 6 full siblings and a paternally related half sibling. The generation to which Case 3 belongs includes many members with the majority under the age at which most gliomas occur.

It is our belief that cases of multiple incidence in a family should be reported in order to contribute, if possible, to understanding of the pathogenesis of this distressing condition. It is our impression, also, that if all such incidences were reported the frequency might prove to be more than coincidence. Koch,[4,5] for instance, presented evidence that led him to believe that there is an hereditary influence on a certain group of gliomas. We have cited, in addition to the cases presented, two other instances of multiple gliomas in a family which have not reached the neurological literature. There may be many more unreported, and the total may be significant of more than coincidence.

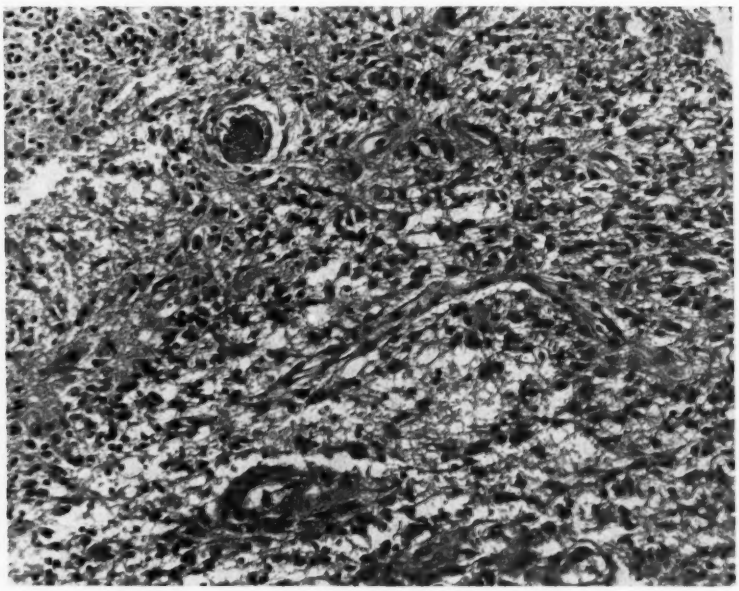

FIG. 1. *Case 2.* Photomicrograph of astrocytoma (grade III).

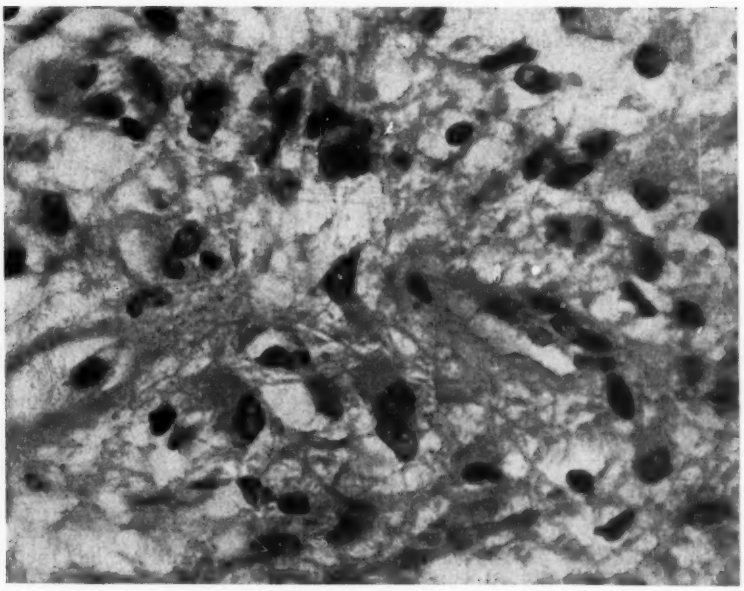

FIG. 2. *Case 2.* High power view of tumor in Fig. 1.

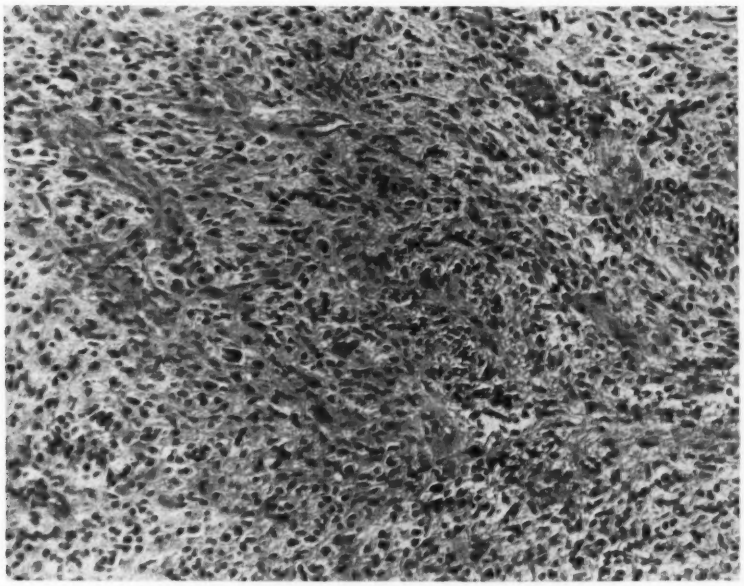

FIG. 3. *Case 3.* Photomicrograph of malignant glioma varying between protoplasmic astrocytoma and glioblastoma multiforme.

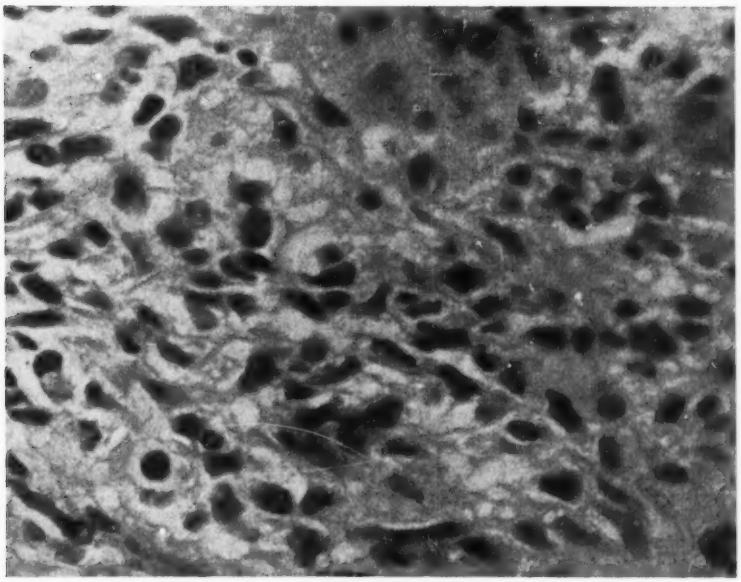

FIG. 4. *Case 3.* Higher magnification of tumor in Fig. 3.

SUMMARY

1. Three cases of gliomas in two generations of a family are described.
2. References to the few accounts of similar multiplicity are cited.
3. Reporting of such multiplicity is suggested as a possible contribution to understanding the pathogenesis of gliomas of the brain.

The authors are greatly indebted to Dr. John R. Green for the material relative to his patient (Case 3), and to Dr. Frank J. Otenasek who kindly made Dr. Dandy's notes (Case 1) available to us.

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MINIATURE STIMULATORS*

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(Received for publication August 10, 1955)

With the increasing availability of miniature electronic components, the practicability of applying their advantages to simplify stimulators has led, in this laboratory, to the development of a number of such instruments. In this paper are described two of these which are now standardized equipment: one is designed primarily for use in animal experiments (by C. G.), the other primarily for the clinical neurosurgeon (by C. M.).

The *animal stimulator* (Fig. 1) is constructed in a small, $2\frac{1}{8}'' \times 1\frac{5}{8}'' \times 4''$ box with three controls: "Single pulse—Off—Repetitive ", "Frequency " and "Voltage." The frequency is adjustable from about 8 to 80 pulses per second and single pulses are also available. The voltage

FIG. 1. Self-contained miniature stimulator with voltage of from 0 to 10 and frequency of 8 to 80 c./sec. The box is $2\frac{1}{8}'' \times 1\frac{5}{8}'' \times 4''$.

is adjustable from 0 to about 10 V.; the wave form is a monophasic square wave, the duration of each pulse depending upon the size of the capacitor selected. The circuit (Fig. 2) is simple and stable, requiring only standard components. A discussion of the circuit appears in the standard texts on transistors.[1]

* Aided by a grant from the National Institute of Neurological Diseases and Blindness, National Institutes of Health, Public Health Service.

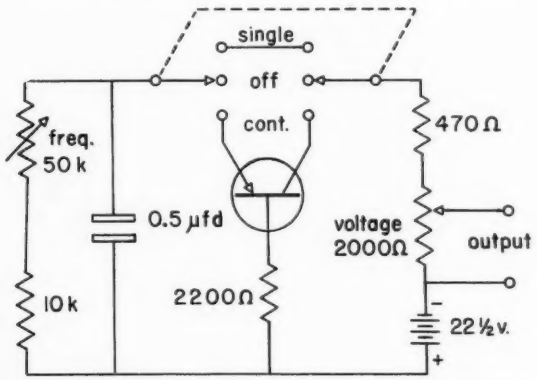

FIG. 2. Circuit of the stimulator shown in Fig. 1. The transistor must be of point contact type, not junction type; the switch must be spring return from the single pulse side.

The *surgical stimulator* is electronically similar to that described above, but it has been altered to fit into a sterilizable shell, small enough to be comfortably held like a pencil. Details of construction were dictated by the following concepts:

1. While the instrument is sterile, it is possible to vary the voltage from the lowest to the highest output.

2. In surgical applications, no precise calibration of intensity is required as the threshold is found by trial.

3. There is no need to alter the frequency or wave form in surgical practice (to be pre-set at between 30 and 60 c./sec.).

4. Though the smallest battery obtainable is capable of 5 hours' uninterrupted duty before the output voltage decreases appreciably, it is desirable, for increased battery life, to interrupt the current flow without contamination.

5. The metal shell is insulated from the electrical circuit to prevent unintentional and extraneous stimulation.

The electronic components and battery fit in a frame (Fig. 3A) which is then enclosed by a thin metal shell (Fig. 3C). This non-sterile unit is dropped, pin end first, into the sterilized shell (Fig. 3D) which is closed off by screwing down the knurled cap (Fig. 3B). Appropriate electrical contacts between inner and outer sections are made without special maneuvers.

Voltages may be varied from 0 to about 10 by turning the indicator ring (Fig. 3h). The battery is turned off by turning the knurled cap counterclockwise for 180°. Four different plug-in electrode arrangements can be used: short bipolar (Fig. 3E), long bipolar, short monopolar, and clip leads.

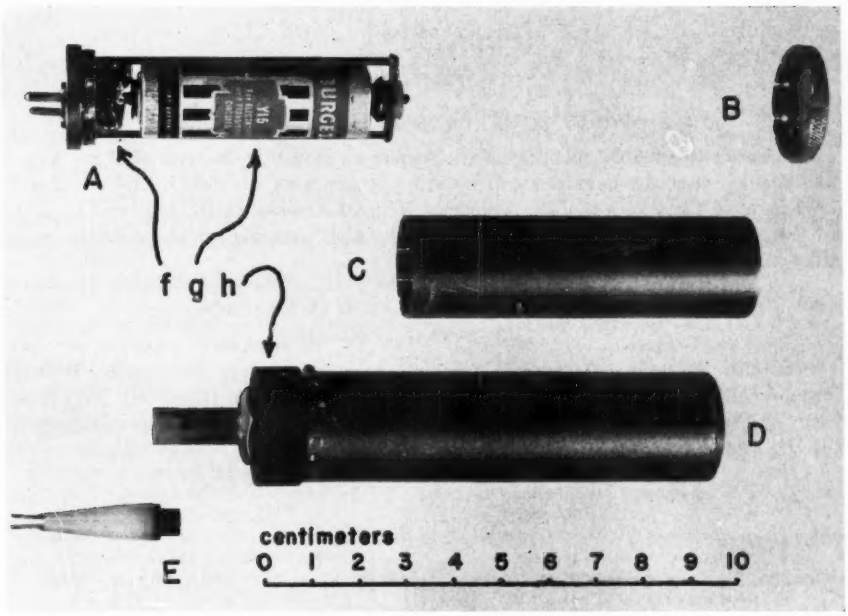

FIG. 3. Sterilizable surgical stimulator. (A) Frame holding the electronic components (except the voltage control) and the battery. (B) Sterile cap which screws onto the outer shell. (C) Inner shell which covers the frame. (D) Sterilizable outer shell. (E) Short bipolar tip. (f) Electronic components. (g) Battery with negative end toward the electronic components. (h) Voltage control indicator ring; the screw head indicates the approximate voltage on the stamped scale.

DISCUSSION

As the maximum potential obtainable from the battery is only $22\frac{1}{2}$ V., electrocution of a patient is not possible. Likewise, the absence of a ground return further protects the patient. Either stimulator may be isolated from ground by a handle or support to diminish the stimulation artifact to a negligible amount. If the battery is exhausted, it may be replaced without contaminating the sterile shell.

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NOTICES

AMERICAN BOARD OF NEUROLOGICAL SURGERY

At the October 1955 meeting of the American Board of Neurological Surgery it was decided that the new five-year training program as outlined in the October 1, 1955 issue of *The Journal of the American Medical Association* (p. 452) will apply to all trainees in neurological surgery who begin their neurosurgical residency on or after January 1, 1956.

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

The 35th annual meeting of the Association for Research in Nervous and Mental Disease will be held on December 9 and 10, 1955 at the Hotel Roosevelt, New York City. The subject of the meeting will be "The Neurologic and Psychiatric Aspects of the Disorders of Aging."

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The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

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